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CHEMICAL WARFARE.[1]

By IVAN MAXWELL, M.D., M.Sc., F.R.A.C.P.,

Member of the Chemical Defence Board, Commonwealth Department of Defence, Melbourne.

THIS lecture is being delivered at the request of the Science Subcommittee of the Victorian Branch of the British Medical Association, and with the approval of Major-General R. M. Downes.

It must be apparent to everyone that it is possible to give only the barest outline of the subject in the course of one lecture. However, an attempt is made to present to you some aspects of the present conception of chemical warfare chiefly from the medical viewpoint. Advantage has been taken of much valuable information contained in the "Medical Manual of Chemical Warfare" issued by the War Office (England).

GENERAL CONSIDERATIONS.

The term "gas" in connexion with warfare is very loosely used, as it includes any chemical substance, whether solid, liquid or true gas, employed for its poisonous or irritant effects on the human body. Thus mustard gas is liquid at ordinary summer temperatures in this country, but it continually gives off vapour—that is, it also exists in the gaseous form. If, however, the temperature should fall below 45° F., as it may in the winter, mustard gas will solidify unless a chemical such as carbon tetrachloride is added to it to lower its freezing point.

Objects to be Achieved by Use of Gas.

The objects to be achieved by the use of gas are as follows: (i) To produce casualties by using gas against personnel. This will be amplified later during the discussion of the properties of individual gases. (ii) To contaminate foods and other stores. Mustard gas vapour readily penetrates fatty substances, hence if butter, cream, fat of meat, or other fat-containing substances are exposed to mustard gas vapour, they may be rendered unfit for consumption. (iii) To compel the use of the respirator. Undoubtedly the wearing of a respirator considerably lowers the physical efficiency of an individual. It is estimated that such loss of efficiency probably amounts to some 25%. (iv) To render ground dangerous to occupy. This particularly applies to the use of persistent gases, such as mustard gas and Lewisite, which vaporize so slowly that they may persist for days, weeks or even months, according to varying meteorological and other conditions. (v) To lower the morale of the civil population. To understand the properties of gases and their limitations and to know thoroughly the methods of effective protection against such gases inspire confidence. Fear of the unknown is an important factor in undermining morale. Hence the necessity for the community gradually to develop an intelligent interest in chemical warfare.

Requirements for Ideal Agents for Chemical Warfare.

Some 3,000 chemical compounds were selected and investigated for offensive or defensive purposes prior to and during the Great War. Of these about thirty were found reasonably suitable, but only about five or six were found to be really successful in actual warfare. The reason for this small number of effective chemicals is that for their successful use in warfare there are many exacting requqirements. These have been ably discussed by Prentiss in his well-known manual "Chemicals in War", and for chemical offensive warfare may be summarized as follows:

Tactical:

1. High toxicity.
2. Multiple effectiveness.
3. Non-persistency.
4. Effects of maximum duration.
5. Immediate effectiveness.
6. Insidiousness in action.
7. Volatility (maximum field concentration).
8. Penetrability.
9. Invisibility.
10. Odourlessness.

[1] An address delivered at a meeting of the Victorian Branch of the British Medical Association on November 13, 1940.

Technical:
11. Availability of raw materials.
12. Ease of manufacture.
13. Chemical stability.
14. Non-hydrolysability.
15. Ability to withstand explosion without decomposition.
16. A solid at ordinary temperature.
17. Melting point above maximum atmospheric temperature.
18. Boiling point as low as possible (if a liquid).
19. High vapour pressure.
20. Vapour density greater than air (the heavier, the better).
21. Specific gravity approximately 1·5.

Let us briefly consider each of these requirements, remembering that for defensive warfare the requirements are in some respects different.

1. High toxicity is an essential property of substances used in chemical warfare. Phosgene is an example of this, for even a concentration of one in 100,000 will soon incapacitate the person who inhales it. Yet not all gases which are highly toxic are effective in warfare. Hydrocyanic acid, although very toxic when inhaled, was a failure in the Great War owing to the fact that being a light gas it very readily diffused into the atmosphere well above the heads of the persons for whom it was intended, and therefore satisfactory concentration of hydrocyanic acid in the field was impossible to obtain.

2. Multiple effectiveness means that the gas should have more than one effect on the individual. Mustard gas not only acts on the skin as a vesicant, but it seriously damages the eyes and also when inhaled causes injury to the respiratory tract. It is multiple in its effect. Phosgene, one the other hand, acts entirely on the respiratory tract and particularly on the alveoli of the lungs.

3. Non-persistence is a necessary quality of an offensive gas, so that when it has done its work and caused casualties among the enemy it will rapidly dissipate, in order that the area may be occupied by advancing troops. Obviously mustard gas, which is very persistent, would not be suitable for use in an area which it was hoped to occupy within a few hours.

4. It is extremely desirable that the casualties produced should be so serious that the stay in hospital will be prolonged.

5. Immediate effectiveness is essential in surprise attacks. Very few gases have this property in field concentrations which are possible to produce. Phosgene is somewhat delayed in its action.

6. Insidious action is a most important attribute. Phosgene can be detected by its smell and its irritant action. Arsine (arseniuretted hydrogen) is completely odourless and non-irritating to the mucous membranes; hence it may be inhaled without the individual's being conscious of its presence in the air. This is probably one of its greatest virtues as an agent in chemical warfare.

7. Volatility is all-important, as the concentration in the atmosphere is largely dependent on this property. Phosgene is very volatile. Mustard gas (used chiefly in defensive warfare) volatilizes so slowly that the concentration in the air remains comparatively low; but this means that the agent persists for a very long time and may cause casualties weeks after it has been disseminated.

8. Penetration through the respirator, or through clothing worn by the civilian or through special protective clothing worn by troops, is a property of so many new gases that this factor alone has been responsible for a complete revolution in the structure of the respirator and in the nature of protective clothing. The modern respirator—of which more later—has been so developed that it affords protection against all known war gases in the concentration which it is found practicable to obtain in the field.

9. Invisibility and (10) odourlessness are two essentials, if the presence of the gas is not to be immediately detected. Chlorine, by the colour of its vapour and its strong odour, so advertises its presence that no person who possesses a respirator is likely to become a victim. Its success in 1915 was due to the fact that the troops upon whom it descended had no respirators and were completely unprotected. Arsine in pure form is both colourless and odourless.

11. Availability of raw material is an absolute essential for the manufacture of chemicals used as war gases. This is so obvious that it need not be further discussed.

12. One of the reasons why comparatively small quantities of mustard gas were used in the Great War was its difficulty of manufacture. Approximately 12,000 tons of mustard gas were used as compared with 100,000 tons of lung irritants (chiefly phosgene), which were easier to manufacture, though relatively not so effective as the vesicants in producing casualties.

13. Chemical stability is essential for material filled into shells. Many bromine compounds attack the iron of the shell wall, and special linings are necessary if such bromine compounds are used.

14. Another necessity is that the substance be non-hydrolyzable. Phosgene readily combines with water and hydrochloric acid is formed, as the following equation shows: $COCl_2 + H_2O = CO_2 + 2HCl$. The hydrochloric acid then slowly dissolves the shell wall and weakens it. Hence great care has to be exercised in filling a shell with phosgene to see that it is perfectly dry.

15. A temperature of 3,000° C. or more may develop when a shell explodes, and this is accompanied by a very high pressure. Many chemicals are completely destroyed by such conditions. Chloropicrin, for example, is decomposed at such high temperatures; but mustard gas and Lewisite are stable.

16. It is convenient to have the "gas" in the solid state; and if this is so the (17) melting point should be above the maximum atmospheric temperature, as the ballistics of the shell may be seriously altered if varying portions of its contents become liquid at the higher temperatures. On the other hand, if the substance is liquid at ordinary temperatures it is desirable that its solidification point should be made as low as possible so that it does not freeze in the cold winter months. Many substances have been added to mustard gas in an attempt to lower its freezing point. The best of these are carbon tetrachloride, chloropicrin and chlorobenzene.

Other desirable qualities of a liquid offensive "gas" are that it should have (18) a low boiling point and hence (19) a high vapour pressure, so that its concentration in the air is at a maximum. Furthermore, the vapour should have (20) a high density so that it keeps close to the ground. The heavier it is in comparison with air, the better, as it persists close to the ground, flows into basements and cellars and does not easily diffuse into the upper layer of the atmosphere above the heads of civilians or combatants.

21. Finally, for uniformity of weight of shells, the material for filling should approximate in specific gravity to 1·5.

The following tabulation illustrates the chief features in which phosgene and mustard gas fell short of ideal requirements:

Phosgene.	*Mustard Gas.*
(i) Lacked multiple effectiveness.	(i) Not easy to manufacture.
(ii) Not immediately effective.	(ii) Not immediately effective.
(iii) Not odourless.	(iii) Not odourless.
(iv) Forms with water vapour a white cloud.	(iv) Low vapour pressure.
(v) Hydrolyzed slowly by water.	(v) Hydrolyzed slowly by water.
(vi) Must be artificially cooled to be filled into projectiles.	(vi) Liquid at summer temperature, solid in winter.

CLASSIFICATION OF GASES.

The classification of gases may now be considered:

Group I (lethal):	(*a*) Vesicants.
	(*b*) Lung irritants.
	(*c*) Paralysants.
	(*d*) Hæmolysants.
Group II (harassing):	(*a*) Lachrymators.
	(*b*) Nasal irritants.
Group III (accidental):	Gases not used as weapons.

GROUP I: LETHAL GASES.

Vesicants.

The best known of the vesicants are mustard gas and Lewisite.

Mustard Gas.

In the crude form such as would be used in chemical warfare, mustard gas—$(ClCH_2CH_2)_2S$—is dark in colour with an odour resembling mustard or garlic, hence its name. It is, of course, not related chemically to mustard. Its boiling point is high (217° C. or 423° F.)—indeed, so high that it vaporizes extremely slowly, hence its persistence even in open spaces. Another physical property of importance is that it freezes at 7° or 8° C. (44° to 45° F.) and hence would be solid in the cold of winter, especially in Europe, unless measures were taken to lower its freezing point, as I have mentioned earlier. The density of mustard gas is higher (1·28) than water; hence it will sink in water unless it falls as very fine droplets on water covered with scum, in which case a considerable amount of the mustard gas may remain on the surface. It is very soluble in animal fats and lipoids, and it therefore penetrates the human skin very rapidly and enters the cells of the body (the cell wall consists largely of lipoid material). Within the cell mustard gas is hydrolyzed and hydrochloric acid is liberated, causing death of the cell. Mustard gas is readily soluble in alcohol, petrol, kerosene, carbon tetrachloride and acetone. It is fairly stable, but is slowly hydrolyzed by cold water and much more rapidly by boiling water. The latter property is made use of in the decontamination of clothing by boiling, thus:

$$S<\begin{matrix}CH_2CH_2Cl\\CH_2CH_2Cl\end{matrix} + 2H_2O = S<\begin{matrix}CH_2CH_2(OH)\\CH_2CH_2(OH)\end{matrix} + 2HCl$$

Mustard gas. Thiodiglycol.

The thiodiglycol formed is non-toxic, and the small amount of hydrochloric acid diluted with much water is harmless to the clothing. Mustard gas has most remarkable powers of penetrating not only the human skin but also clothing. Ordinary clothing gives very little protection from liquid mustard gas, and sacking such as that used as containers for wheat or wool bales is immediately penetrated; even leather gloves are not impervious to the liquid. Rubber and oilskin have more resistance, but even they are not by any means completely protective; it is only a question of some hours and the liquid will pass through them. The surface of glazed porcelain, for example, crockery, is completely resistant; but mustard gas readily enters concrete or woodwork, and if a person sat on an object so contaminated the warmth of the body would cause the mustard gas to vaporize and penetrate the clothing, and cause injury to the skin beneath. It readily soaks into bitumen roads, which then become very difficult to decontaminate.

Toxicity of Mustard Gas.—Mustard gas is not a general systemic poison. When persons succumb to its effect, the cause of death is secondary infection with a variety of microorganisms. It is estimated that if 20 milligrammes of mustard gas vapour were quickly inhaled into the lungs, it would cause such damage to the tissue locally that death would ensue. A calculation on this basis indicates that one ton would be sufficient to kill 45,000,000 people. Actually 12,000 tons used in the Great War were responsible for approximately 400,000 casualties. This indicates that relatively little of the gas was absorbed by the troops in actual warfare. Much investigation in the last twenty years has been devoted to determining more effective methods of distributing mustard gas so that full advantage might be taken of its toxic properties. Owing to the fact that mustard gas has toxic effects in a concentration that is barely perceptible to the sense of smell, it is particularly dangerous in low concentrations, as contamination may be unsuspected and its insidious actions may continue quite undetected by the victim. Its action is delayed, symptoms not appearing for some hours after contamination. The respirator is completely protective against mustard gas.

Lesions Produced by Mustard Gas.—Lesions produced by mustard gas may be considered under two headings: (i) liquid mustard gas, (*a*) skin burns, (*b*) eye burns, and (ii) mustard gas vapour, (*a*) action on the skin, (*b*) action on the eyes, (*c*) action on the respiratory tract and (*d*) other effects of a minor nature. Droplets of liquid mustard gas falling upon the skin will penetrate it within two or three minutes, hence the necessity of immediate measures to combat its effect. A vesicle will form containing yellow serum, which later may become infected. Around the vesicle is an area of erythema. These vesicles may coalesce if they are numerous. Droplets falling into the eyes will not give rise to immediate symptoms; but in half an hour or so the eyes may begin to swell and later may become very œdematous and painful, conjunctivitis, lachrymation, photophobia and blepharospasm being the chief features. If, therefore, measures are not promptly adopted for relief of the condition, the eyesight may be lost. The action of mustard gas vapour on the skin is most pronounced in the moist parts of the body, such as the axillæ and perineum, but it also affects the exposed parts of the body. The reaction varies from mild erythema to intense erythema with vesiculation, depending on the concentration of vapour in contact with the body and on the duration of its action. The effect of vapour on the eyes is similar to that of the droplets, but less intense, and seldom leads to irreparable damage. In the respiratory tract the irritant action of the vapour causes rhinitis, laryngitis, tracheitis and bronchitis, and in some instances bronchopneumonia ensues, due to secondary infection with microorganisms. The chief cause of death is bronchopneumonia. Pulmonary œdema is not a prominent feature of the action of mustard gas vapour, as is the case when phosgene is inhaled.

Method of Dispersal of Mustard Gas.—As has been stated, since the Great War, much research has been done on the problem of the most effective manner of using mustard gas. The methods of dispersion may be summarized as: (i) spray from aeroplanes, (ii) bombs from aeroplanes, (iii) shells, (iv) spray from specially constructed vehicles. It is likely that the first and second methods of dispersion would prove most effective. The amount contained in a shell compared with the total weight is small, owing to the fact that the shell wall must be strong, whereas in a bomb the relative amount of mustard gas is much greater, owing to the thin wall of the bomb. Ordinary mustard gas can be altered in its viscosity by the addition of suitable chemicals, so that the droplets when falling from an aeroplane do not shatter. If excessive shattering of the droplets occurred then they would become too fine to have satisfactory penetrating power after falling on clothing. The persistence of the gas after its distribution depends upon various meteorological conditions, such as wind, rain, temperature of the air, atmospheric pressure, clouds and humidity. The detection of mustard gas is either by the sense of smell, which is by far the most effective method, or by certain chemical detectors which change from yellow to red when drops of liquid mustard gas fall on them. Protection against liquid and mustard gas vapour is difficult. Without the specifying of details, this consists in the wearing of a respirator, a protective hood or helmet, an oilskin jacket and trousers, rubber boots and rubber or oilskin gloves. This protective clothing prevents prolonged physical activity, and hence for troops special capes, eye shields and clothing that is impregnated with a chemical to destroy gas are used and the respirator is carried for immediate use. This allows much greater freedom of movement.

Treatment of Personnel Contaminated by Mustard Gas.—Treatment of personnel contaminated by mustard gas may be summarized under two headings, preventive and curative. Preventive treatment consists in (*a*) bleach treatment, (*b*) the application of anti-gas ointment number 2, (*c*) swabbing with a solvent of liquid mustard gas, and (*d*) scrubbing with soap and water. Bleach is a synonym for bleaching powder or chloride of lime. It may be used in aqueous solution, equal quantities of water and supertropical bleach being mixed to form a paste. If this is rapidly applied after contamination and then removed after a few minutes by washing off with water, it is usually quite effective. It must not be left on the skin for any length of time, as it will produce irritation. In the form of an ointment it is useful, but again must not be

allowed to remain on the skin for very long. The ointment is composed of equal parts of supertropical bleach and petroleum jelly. The action of bleaching powder may be indicated in the following equation:

$$CaOCl_2 + S<\frac{CH_2CH_2Cl}{CH_2CH_2Cl} = SO<\frac{CH_2CH_2Cl}{CH_2CH_2Cl} + CaCl_2$$

Bleaching powder.	Mustard gas.	Mustard sulphoxide.	Calcium chloride.

Mustard sulphoxide which is formed is harmless. Bleaching powder as paste or ointment must not be allowed to enter the eyes. A much more effective ointment is anti-gas onitment number 2, the composition of which is secret. It can be allowed to remain on the skin without danger of irritation and can be used as a prophylactic, the ointment being rubbed into the exposed parts in anticipation of a gas attack. Swabbing with petrol, kerosene, carbon tetrachloride or other solvents of liquid mustard gas is a very effective way of removing droplets from the skin. It must be remembered, however, that this merely removes the mustard gas and does not destroy it. The cloth used for swabbing must be destroyed as soon as possible. Care must be taken that the solvent is not inadvertently allowed to run over the skin and thus spread the mustard gas to other parts of the body. Scrubbing the body with soap and water, preferably under a shower, may mechanically remove the mustard gas in the lather, but does not destroy it, as cold water causes very slight hydrolysis of the gas. Each of these prophylactic measures is effective if carried out carefully, and the one selected for use depends upon which is available.

With regard to curative treatment, the eyes may be discussed first. The fluid for lavage of the eyes may be water, normal saline solution, a 2% solution of sodium bicarbonate or a saturated solution of boric acid. Immediate and repeated lavage is most essential. Every moment that passes after droplets of mustard gas have entered the eyes renders successful treatment more difficult. Paraffin is used to prevent the eyelids from adhering, and atropine as a 1% ointment relieves pain. Cocaine is contraindicated, as it interferes with the successful healing of the eyes. For rhinitis, the nose should be irrigated with normal saline solution. For laryngitis, inhalations of *Tinctura Benzoini Composita* are used.

The treatment of bronchopneumonia follows the usual symptomatic methods. It is not yet known whether the sulphanilamides will be helpful in combating this condition if mustard gas is used in the present war. Slight erythema of the skin is treated with a simple dusting powder of chalk, starch, boric acid and zinc oxide. Blisters should be evacuated and the outer dead skin removed, and lint soaked in a solution of 20% "Dettol" in 5% tannic acid should be applied for ten or twelve hours. By that time the tannic acid has precipitated the protein and formed a firm layer on the surface. This can be further sprayed with tannic acid solution.

Tannic acid as a 2·5% solution with the addition of 0·5% of phenol may also be used. Sometimes instead of the aqueous solution it is used as a jelly, as in the form of "Tannafax". The area around the burns should be swabbed with antiseptic solution to reduce the tendency to infection. When the condition is septic, continuous baths of a mildly antiseptic character at body temperature may be valuable.

Invalidism after Mustard Gas Poisoning.—The experience of the problem of invalidism, due to mustard gas poisoning in and after the Great War, is indicated by the following quotation from the "Manual of Chemical Warfare":

> The ultimate invalidism from mustard gas vapour was . . . very small. As in phosgene poisoning it is probable that any persistent chest trouble was due to mischief wrought by the smouldering inflammation of secondary bacterial infections rather than by a direct chemical action. . . . Some cases showed a tendency to relapses of bronchitis but there was no special proneness to pulmonary tuberculosis. . . . Doctors should realise that gas poisoning, whatever the chemical irritant concerned, does not in itself cause a permanent poisoning of the patient or chronic impairment of his health. It is necessary to insist on this truth lest the patient be allowed to develop a morbid dread and drift into neurasthenia and general debility.

Functional disorders were not uncommon. Of the eye casualties caused by mustard gas, 75% could be classed as mild, and the patients were fit for duty again in two weeks. Cases of intermediate intensity represented 15%, and the patients recovered in four to six weeks; whilst in the remaining 10%, which were regarded as severe cases, two to four months were taken for recovery and some patients even lost their eyesight. Attention is drawn to functional disturbances, such as photophobia, persisting long after the organic basis for this condition had entirely disappeared. It is emphasized, for example, that the use of eye shades for an unnecessarily long time tends to perpetuate functional photophobia.

Lewisite.

Lewisite, though differing chemically in structure from mustard gas and containing arsenic, nevertheless has many properties in common with mustard gas. It is a liquid, dark in colour and with an odour resembling geraniums. It boils at 190° C. (374° F.), and hence vaporizes slowly and is a very persistent gas. Its freezing point (–13° C. or 8·6° F.) is much lower than that of mustard gas, and so there is little tendency for it to freeze in the winter months. Its solubilities are similar to mustard gas, but it is less stable.

Toxicity.—Lewisite is an eye irritant, lung irritant and vesicant, and it also acts as a general systemic poison in virtue of the arsenic which it contains. A comparison of Lewisite and mustard gas is seen in the following tabulation:

Lewisite.	*Mustard Gas.*
(i) Contains arsenic.	(i) Contains no arsenic.
(ii) May cause systemic poisoning.	(ii) Does not cause systemic poisoning.
(iii) Rapidly hydrolyzed by water.	(iii) Slowly hydrolyzed by water.
(iv) Moderately stable.	(iv) Very stable.
(v) Moderately persistent.	(v) Very persistent.
(vi) Vapour causes immediate irritation.	(vi) Vapour causes delayed irritation.
(vii) Liquid causes immediate tingling or pain.	(vii) Liquid causes delayed tingling or pain.

In a brief consideration of the nature of casualties from Lewisite, attention should be first drawn to the fact that Lewisite, both in the liquid form and as a vapour, acts immediately as an irritant and so betrays its presence. Liquid entering the eyes causes immediate pain, and on the skin it penetrates almost instantly, with the production of irritation. This should be contrasted with the delayed development of signs and symptoms in the case of mustard gas. There is very little erythema around the vesicle formed on the skin. The fluid content of the vesicles contains arsenic. If 0·3 cubic centimetre in the form of droplets falls upon the skin and is absorbed, it is sufficient to cause arsenical poisoning. Further features of interest concerning Lewisite are that small drops are destroyed by wet clothing, and hence would be relatively ineffective as a spray in wet weather. The general treatment, both preventive and curative, of Lewisite casualties is similar to that of mustard gas; but it should be noted that bleach ointment is ineffective in combating Lewisite. Bleach paste is, however, quite satisfactory, and ointment number 2 is also very effective.

Lung Irritant or "Choking" Gases.

The following are the chief members of the lung irritant group: (i) chlorine, a greenish-yellow cloud, with a smell similar to bleaching powder; (ii) chloropicrin, a yellow liquid, which boils at 233·6° F., has a smell like that of chlorine and is a powerful lachrymator; (iii) phosgene, a liquid which boils at 46·8° F., is a colourless gas and has a smell of musty hay; (iv) di-phosgene, a liquid which boils at 262·4° F., and has a smell like that of phosgene.

Phosgene.

Phosgene will be taken as a typical representative of the lung irritant or "choking" gases. It is a very volatile liquid, boiling at 46·8° F., and when

disseminated in the air it can be detected partly by its smell of new-mown hay and partly by slight irritation of the mucous membranes with which it comes in contact. When inhaled, its chief action is to cause pulmonary œdema. The blood becomes more concentrated and thrombosis is likely to occur in small vessels within the lungs. Much coughing is induced, and rupture of pulmonary alveoli is common. A small amount of pleural effusion may occur. In mild cases the face may be flushed and the respirations rapid, and a painful cough may be present. In more severe cases there is "blue" cyanosis of the face, but the pulse may still be full and strong. In the most severe cases there is leaden "grey" cyanosis, with a very rapid and feeble pulse and rapid respirations. Physical examination of the lungs gives little indication of the gravity of the case, and the prognosis is determined chiefly by the colour, the nature of the pulse, and the character of the respirations.

The treatment of phosgene poisoning may be summarized as follows: (i) rest, (ii) warmth, (iii) venesection, (iv) the administration of oxygen, (v) suitable drugs, and (vi) diet. Rest is most important, as undue exertion on the part of the patient may cause collapse and death. Warmth is necessary, as the patient is in a state comparable to shock. If the colour is of the "blue" cyanosed type, then venesection (removal of 400 to 600 cubic centimetres) may be beneficial; but venesection is contraindicated if leaden "grey" cyanosis is present. The most important treatment is the administration of oxygen by nasal catheters, by oxygen tent or by a Haldane or similar type of inhalation apparatus. From five to ten litres per minute are required, and the administration must be continuous for as long as the patient is in danger. No drugs are of special value in the treatment of phosgene poisoning. Atropine does not stop the tendency to pulmonary œdema. A simple diet is allowed, and drinks are given freely to replace the fluid that is lost from the circulation. Sequelæ are not common or serious. The respirator is completely protective against phosgene. Since phosgene can be detected by its smell and slight irritating action on the respiratory tract, the greatest danger lies in the first couple of minutes of its inhalation, when a person taken by surprise may inspire a highly toxic amount before being able to adjust his respirator. After the first few minutes the warning to those downwind will be in ample time to prevent casualties.

Paralysant Gases.

The chief members of the paralysant group are hydrocyanic acid and sulphuretted hydrogen.

Hydrocyanic Acid.

Hydrocyanic acid is a colourless liquid which boils at 78° F. It is very volatile and lighter than air, so that in open warfare it loses much of its effectiveness by rapid diffusion. It inhibits oxidation in animal tissues by acting on the cytochrome-indophenol oxidase complex which controls cellular respiration, and in strong concentration rapidly paralyses the respiratory centre. The symptoms with strong concentrations are palpitation, hurried breathing, rapid development of coma and death. Lesser concentrations in the air cause headache, giddiness and nausea. The ordinary charcoal respirator is not very effective, and for satisfactory protection special pads soaked in caustic soda are necessary in the container. The treatment of hydrocyanic acid poisoning is immediate artificial respiration, accompanied, if possible, with the use of "Carbogen". Various chemicals, such as methylene blue, sodium thiosulphate and glucose, have been used to combat poisoning by this agent, but they have not been very successful. Its use in modern warfare is unlikely, to judge by its failure in the Great War.

Sulphuretted Hydrogen.

Sulphuretted hydrogen is a colourless gas which is heavier than air. It may be found in tunnels, dugouts and other confined spaces, where it may be detected partly by the sense of smell and also by use of lead acetate papers. On inhalation it causes irritation of the eyes and the respiratory tract, hurried respiration, pallor, and (if in sufficient concentration) coma followed by respiratory failure. The respirator is protective. The treatment for poisoning by this gas is artificial respiration, if possible combined with the inhalation of "Carbogen".

The Respirator and Paralysant Gases.

Before I leave this group of paralysant gases, reference should be made to the fact that hydrocyanic acid will act on the metallic catalyst which is present in the charcoal of the respirator to form metallic cyanides, and in this way may destroy the respirator's protective power against arsine. It is very doubtful, however, whether sufficient concentration of hydrocyanic acid could be obtained in the field to cause serious formation of metallic cyanides in the respirator.

Hæmolysants.

Arseniuretted Hydrogen (Arsine).

Arseniuretted hydrogen, also known as arsine, is a colourless inflammable gas. It has approximately the same toxicity as phosgene, but being odourless and non-irritating to the respiratory tract, it is not detected in the air by the ordinary or special senses as is phosgene. Arsine is liberated from metallic arsenides, such as calcium arsenide, by the action of water. If calcium arsenide was distributed as a fine powder on the ground and was moistened by rain or dew, then arsine would be liberated. Other methods of distributing arsine are available, but I am not at liberty to discuss these.

Toxicology.—Arsine when inhaled passes into the blood, and has a very great affinity for red blood corpuscles, in which it becomes strongly concentrated. The fragility of the corpuscles is increased and hæmolysis occurs, the free hæmoglobin liberated into the plasma being excreted by the kidneys, causing hæmoglobinuria or methæmoglobinuria. Some of the hæmoglobin diffuses into the tissue spaces, and a portion of it is converted into bilirubin, causing the person to become jaundiced. Tubular nephrosis is a prominent feature in the kidneys, and is accompanied by albuminuria and the formation of casts. The blood urea percentage may be raised. Pronounced congestion and yellow areas of necrosis are found in the liver, the spleen is enlarged and dark in colour, but the heart and lungs are apparently not damaged by arsine.

Signs and Symptoms.—The initial symptoms are headache, giddiness and perhaps vomiting, followed by dyspnœa. Later jaundice may occur, associated with hæmoglobinuria and methæmoglobinuria. About two milligrammes per kilogram of body weight are a lethal dose, and half that quantity may cause severe hæmoglobinuria.

Detection of Arsine.—Arsine, being odourless, colourless and non-irritating, cannot be detected by the ordinary or special senses. Detection in the air is based upon chemical methods. Certain test papers will show a yellow or orange colour in the presence of arsine. For field purposes one can determine the degree of hæmolysis caused by arsine in the following way.

Take three drops of blood and mix with 12 drops of 3·8% sodium citrate solution. Draw this mixture into a tube of two or three millimetres' bore and 20 centimetres long. Let it stand for half an hour in a vertical position. Hæmolysis can be seen by the colour of the plasma. The degree of hæmolysis can be estimated by comparison with a series of standards.

The modern respirator containing a metallic catalyst in the charcoal is completely protective against arsine.

Treatment.—The forms of treatment may be summarized under the following headings: (i) blood transfusion, (ii) diuretics, (iii) alkalis, (iv) glucose, (v) oxygen, (vi) treatment in convalescence.

(i) Blood transfusion is by far the most important form of treatment. Red blood corpuscles are being destroyed and hæmoglobin is rapidly lost from the body, so that it is only logical to replace the corpuscles as quickly as possible. Small transfusions (500 cubic centimetres) repeated as required are recommended. The transfused blood is not hæmolysed. There is no experimental evidence in favour of bleeding the patient to remove the arsine. (ii) The best diuretic is water, and four or more pints should be given per day. Water probably washes the casts out of the renal tubules and in this way restores renal efficiency. (iii) Alkalis in the form of potassium citrate or sodium

citrate are given in sufficient quantity (30 to 60 grains) every four hours to keep the urine alkaline. If the urine is allowed to become acid it may precipitate the hæmoglobin as acid hæmatin in the tubules of the kidneys, and so impair renal function as to induce uræmia. (iv) Glucose is administered freely so as to restore the glycogen content of the damaged liver. Hepatic efficiency is largely dependent on a satisfactory glycogen content. (v) It is doubtful whether oxygen is of much value. The hæmoglobin in the corpuscles that are not destroyed is fully saturated, and the administration of oxygen could be beneficial only by the oxygen's being carried in slight excess in the plasma; but this is a minor factor. (vi) The treatment in convalescence should include the administration of liver extracts, as the hæmatinic principle may not be satisfactorily stored in the damaged liver.

GROUP II: HARASSING AGENTS.

The harassing agents are lachrymators (tear gases) and nose irritants. The lachrymators are ethyl-iodo-acetate (K.S.K.), bromo-benzyl-cyanide (B.B.C.) and chloro-aceto-phenone (C.A.P.); the nasal irritants (nose gases) are di-phenyl-chlor-arsine (D.A.), di-phenyl-amine-chlor-arsine (D.M.) and di-phenyl-cyano-arsine (D.C.).

Lachrymators.

Many compounds, both liquid and solid, are lachrymators. Liquid lachrymators may be dispersed by shell or bomb or by mechanical spraying. The solid C.A.P. is dispersed by heat or sprayed in solution, but is not suitable for shell. The effect of lachrymators is to cause watering of the eyes and spasm of the eyelids, and a burning feeling in the throat and chest. The action is temporary and soon disappears if the person puts on the respirator or enters a gas-proof shelter. The respirator is completely protective. If liquid enters the eyes—owing to aerial spray or bursting bombs—lavage of the eyes with normal saline solution or water should be carried out immediately.

Nasal Irritants.

The chief nasal irritants or sternutators are solid organic arsenical compounds. They are usually referred to as toxic smokes, and were designated "blue cross" by the Germans in the last war, whilst mustard gas was referred to as "yellow cross", and lung irritants were known as "green cross". When inhaled, they cause acute pain in the nose and nasal sinuses, fullness in the head and sneezing. Acute mental disturbance may accompany these symptoms. Treatment may be unnecessary; but the inhalation of a little chloroform, or local irrigation and gargling with a 5% sodium bicarbonate solution may give symptomatic relief.

The problem of protection against toxic smokes involves the use of a special filter in the respirator, and this will be referred to when respirators are being discussed.

GROUP III: OTHER DANGEROUS GASES ENCOUNTERED UNDER WAR CONDITIONS.

Dangerous gases which may accidentally be encountered under war conditions are carbon monoxide, nitrous fumes and screening smokes; the screening smokes are phosphorus, titanium tetrachloride, stannic chloride, chlorosulphonic acid and oleum.

Carbon Monoxide.

Carbon monoxide may be encountered in burning buildings, broken gas mains, under-water explosions, pill boxes, turrets, gun emplacements and within tanks. It will be remembered that carbon monoxide when inhaled combines with hæmoglobin (its affinity for hæmoglobin is 300 times the affinity of oxygen for hæmoglobin) to form carboxyhæmoglobin, and the person may suffer from severe anoxæmia. Death usually occurs when the carboxy-hæmoglobin is 70% of the total circulating blood pigment.

Unfortunately the ordinary service respirator is not protective against carbon monoxide, so that if a person has to enter a space suspected of being contaminated with carbon monoxide a special oxygen breathing apparatus should be worn.

Artificial respiration and also the free use of "Carbogen" form the treatment of carbon monoxide poisoning. "Carbogen" is preferable to oxygen, as the carbon dioxide stimulates the depth of respiration and enables the partial pressure of oxygen in the alveoli to be raised.

Before one leaves the subject of carbon monoxide poisoning, reference must be made to the metallic carbonyls. Iron and nicol carbonyl, to mention only two, will act as poisons when inhaled. The modern respirator will give protection against carbonyls as such, but the carbonyl when adsorbed on the charcoal in the respirator proceeds to liberate carbon monoxide, which is then inhaled. Fortunately the experimental evidence so far suggests that the maximum field concentration of the carbonyl is not likely to be sufficiently high to cause enough carbon monoxide to be inhaled from the decomposition of carbonyl in the charcoal of the respirator to cause danger to life.

Nitrous Fumes.

Nitrous fumes are mixtures of oxides of nitrogen due to incomplete combustion of nitro-explosives—for example, cordite. These fumes may accumulate in magazines of ships, during tunnelling operations, in gun pits, in armoured cars and in tanks. They react with water to form nitric and nitrous acids. The nitric and nitrous acids which are found in the bronchial tubes when the fumes are inhaled cause considerable irritation, and pulmonary œdema may ensue. Furthermore, alkaline nitrites are formed in the respiratory tract by interaction with alkaline secretions. These nitrites when absorbed tend to lower blood pressure.

The ordinary respirator gives very limited protection against nitrous fumes. Furthermore, carbon monoxide usually is present with the nitrous fumes and the respirator gives no protection against this gas. An oxygen breathing apparatus must be worn in an atmosphere contaminated with these gases. Treatment follows that outlined for phosgene poisoning.

Screening Smokes.

Screening smokes in concentrations which are effective as screens are non-toxic, and troops can operate in them without wearing respirators. The chemicals which are used to produce these screens are all corrosive or dangerous to handle. Those working with such substances should wear goggles or respirators, gloves and sometimes special protective clothing.

Phosphorus.

Phosphorus, when exposed to the air, rapidly oxidizes to form the fumes of phosphorus pentoxide, and is used as a smoke screen. If fragments of phosphorus fall upon the skin they will cause severe burns. The part should be immediately immersed in water and the phosphorus scraped off or covered with a thick pad soaked in water. Immersion of the part in a 1% or 2% solution of copper sulphate causes the formation of an inactive phosphorus compound.

Titanium Tetrachloride.

Titanium tetrachloride is a yellow corrosive fluid which, when in damp air, gives off a dense white cloud: $TiCl_4 + 4H_2O = Ti(OH)_4 + 4HCl$. It is used in the laying of smoke screens from aeroplanes. Contamination of the skin and eyes with the liquid is treated by lavage with water and the use of a sodium bicarbonate solution.

Stannic Chloride.

Stannic chloride is a fuming corrosive liquid which produces a white cloud on contact with air. The treatment of splashes due to the liquid is similar to that of titanium tetrachloride.

Chlorosulphonic Acid.

Chlorosulphonic acid is a highly corrosive liquid which on contact with moisture gives off a dense white cloud. With water it develops great heat, and the acid may be scattered about. Protective goggles or respirator and oilskin coat, rubber gloves and rubber boots should be worn by persons handling this acid. Contamination of the eye or skin should be treated by free lavage with water followed by sodium bicarbonate solution.

Oleum.

Oleum is a very corrosive liquid, consisting of sulphuric acid and containing sulphur trioxide. It gives off white fumes. Protection is as for chlorosulphonic acid and the treatment for contamination is similar.

THE RESPIRATOR.

The respirator consists of a canister or container in which are special materials, the function of which it is to prevent the access of deleterious material to the respiratory tract. Briefly, these consist of charcoal and certain filtering pads. The power of adsorption of the noxious gases is all-important. This adsorptive power varies very considerably with the source of charcoal. That, for example, obtained from coconut is very much more effective than that from cedar. By a special method of treatment the charcoal is activated—that is, it becomes more porous in character and has its adsorptive powers greatly increased. Most gases, with the exception of carbon monoxide and nitrous fumes, are well adsorbed by the charcoal in the container. However, another notable exception is arsine. An ordinary charcoal respirator is not effective against this gas. Fortunately, if the charcoal is treated with certain metallic salts, the metal retained by the charcoal acts as a catalyst for the oxidation of the arsine, and thus the modern respirator is protective against this gas. In the German respirator copper or manganese is used as the metallic catalyst. An even more effective metal is used in the British respirator.

The toxic smokes, such as diphenylchlorasine (D.A.), are in the form of particulate matter and are not adsorbed by charcoal, so a filter has to be used to remove the particles from the impure air. The exact nature of this filter is secret, and its mode of action is complex, depending upon varying physical factors. Suffice it to say that before the service respirator is distributed to the troops these filters have to pass a severe test to indicate their filtering power against toxic smokes. The face-piece is so constructed that it fits the face accurately and prevents air from entering the bronchial tubes by any path other than through the container of the respirator. The face-piece is connected to the container by corrugated tubing. The German respirator has the container attached directly to the face-piece, and hence the weight hanging from the head is very much greater than in the British respirator.

SOME STATISTICS.

At the beginning of the Great War (1914) Germany controlled approximately 86% of the dye industry with which the manufacture of "gas" is so closely linked, whilst Great Britain's corresponding figure was only 2·5% and that of the United States of America was 1·8%. Great progress has been made since then in the dye industry in Great Britain and in the United States of America, as the following figures show (Table I).

TABLE I.

Production of Dyes. (After Prentiss.)

Year.	Germany. (Pounds.)	Great Britain. (Pounds.)	U.S.A. (Pounds.)
1913	308,560,000	9,111,000	6,612,000
1933	145,000,000	52,945,000	100,953,000

In the Great War, Germany is credited with having used 10,000 tons of vesicants (mustard gas almost entirely), France 2,140 tons and Great Britain only 100 tons. Of lung irritants, Germany used 48,000 tons, France 34,000 tons, and England 23,000 tons. It is likely that if chemical warfare is adopted in this present conflict, vesicants will play a very prominent part, since a casualty was produced in the Great War for every 60 pounds of vesicant used, whilst 230 pounds of lung irritant gas were required to produce such casualty. The actual number of deaths caused by gas compared with non-fatal injuries caused by gas varied from about 3% to 9% in the armies of different countries (the United States of America excluded). The deaths due to high explosives and forms of warfare other than chemical warfare represented 25% or more of the total casualties. It is estimated that approximately 1,296,000 casualties due to chemical warfare occurred in the Great War. Of this number 91,000 were fatal and 1,205,000 were injuries from which the troops recovered. The total number of deaths for all countries involved and from all causes during the Great War was about 8,000,000, whilst the wounded (deaths excluded) numbered approximately 21,000,000.

PROTECTION AGAINST GAS.

Protection against gas may be either individual or collective. Protective clothing, the respirator and protective ointment have already been discussed. A few words may now be added concerning the eyeshield and the anti-gas shelter. The eye shield (of simple structure) is to protect the eyes from droplets of corrosive fluid falling through the air from sprays directed from aeroplanes. Such droplets may be liquid mustard gas, Lewisite, corrosive acids, such as hydrofluoric, sulphuric or nitric, or other corrosive fluids. Eye shields would be a very important part of the equipment of the individual if spray from aeroplanes was used.

Air-raid shelters or anti-gas shelters may be (*a*) large compartments ventilated by filtration units, or (*b*) rooms in private homes made relatively gas-proof and not ventilated. Only this latter non-ventilated room will be discussed. It will be the duty of most civilians during a gas attack to return to their gas-proof room and remain there until the all-clear signal is given. Lay persons are apt to think that it would be impossible to remain in a room for any length of time when access to external air is cut off. Physiologists have shown, however, that in such a room a gradual rise in carbon dioxide content to 3% is permissible, while the oxygen content will fall to between 17% and 18%. Normal air contains about 0·04% of carbon dioxide and nearly 21% of oxygen. Such a fall in the oxygen content of the air breathed will have no effect on the depth or frequency of respiration, but the increase in the carbon dioxide content of the air from 0·04% to about 3% will increase the depth and to a less extent the frequency of respiration. Headache and some degree of lassitude may be experienced, but nothing more serious. The wet bulb temperature should not be above 80° F. The question arises as to the size of the room necessary to give sufficient air space per person in such conditions. This is expressed in square feet of surface. If the total surface area of the room—that is, of the walls, floor and ceiling—is calculated in square feet, then a 100 square feet of surface for each person should be adequate for twelve hours' occupation in the climatic conditions found in England and in the milder portions of Australia. Thus a room the dimensions of which are 10 feet by 10 feet by 8 feet has a surface area of 520 square feet, and the number of occupants allowed for twelve hours is five persons. By the end of this time the carbon dioxide content of the room may be approaching 3% and the oxygen content would have diminished almost proportionately, but the occupants should still be quite well.

CONCLUSION.

Many volumes have been written on the subject of chemical warfare. This brief summary has been made not only to refresh the memory of those to whom perhaps its details have once been only too familiar, but also to enlighten others who may, if they so desire, find further information in the works included in the accompanying short bibliography.

Finally, it should be emphasized that much of the latest information from chemical warfare experimental stations in England, which comes direct to military headquarters in Australia, is not available to the general public.

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SOME OBSERVATIONS ON THE BIOCHEMICAL CHANGES ASSOCIATED WITH HÆMORRHAGE FROM THE STOMACH AND DUODENUM.[1]

By MARJORIE BICK, M.Sc.,

AND

IAN J. WOOD, M.D., M.R.C.P., F.R.A.C.P.,

Marion Carty Research Scholar; Physician to Out-Patients, Royal Melbourne Hospital.

(From the Walter and Eliza Hall Institute of Research in Pathology and Medicine.)

IN 1936 one of us (I.J.W.[1]) made a study of the treatment of severe hæmorrhage and paid special attention to the rise in the blood urea level in cases of hæmatemesis. During the past two years a more detailed investigation has been undertaken of the biochemical changes which occur as the result of massive hæmorrhage from the stomach or duodenum. Eight patients have been studied in detail and occasional determinations have been made in five other instances.

In the cases studied in detail, determinations were made of the pulse rate, the blood pressure, the hæmoglobin value, the blood urea, sugar and chloride levels, the plasma protein content, the serum calcium content, and of the urinary volume and chloride, glucose, urea and calcium contents. In addition, observations were made on the caloric intake by mouth and on the effects of the intravenous infusion of blood and of glucose saline solution. The same biochemical observations were made on two normal controls over a period of three days. They were placed at rest and given the standard diet and intake of fluid (Figure IV, Tables IIIA and IIIB). The blood volume was determined in one subject and in one control.

It was found that after a hæmorrhage the blood pressure was usually low, but slowly rose again after the bleeding had ceased. This rise was hastened by blood transfusion. The hæmoglobin value was low in all cases, falling both during and immediately after a hæmorrhage. It was a valuable guide to the patient's progress. The plasma protein content was lowered in all cases, but usually only to a minor degree; occasionally a low level was reached. The blood urea content was elevated in all cases and appeared to be a good index of the general condition of the patient; it was highest when the patient was not only exsanguinated, but was drowsy, thirsty and dehydrated. The blood chloride content was always low, and this finding was associated with a pronounced diminution of chloride in the urine. The liberal administration of chloride by mouth, rectally or intravenously, appeared to benefit the patient. Immediately after a massive hæmatemesis, when the patient was first admitted to hospital, the urinary output was usually diminished; but it soon improved with the liberal administration of fluid. To maintain a satisfactory positive fluid balance the intravenous injection of blood or glucose saline solution was sometimes necessary. With regard to diet, it was often difficult to entice patients to take an adequate number of Calories, owing to a pronounced distaste for food. Fluid, especially plain cold water, was taken more eagerly. There was often a tendency to acidosis, as shown by a diminution in the alkali reserve. The serum calcium content was usually maintained within normal limits.

In the present series transfusion of citrated blood produced an elevation in the blood pressure and hæmoglobin percentage. There was also a slight rise in the plasma chloride and protein levels; the blood urea and serum calcium levels tended to fall.

The intravenous injection of glucose saline solution increased both the plasma chloride content and the output of chloride in the urine.

[1] This work was carried out with the aid of a grant from the National Health and Medical Research Council.

The methods employed in the biochemical analyses will be described first, and this description will be followed by a detailed account of the results of our investigations.

Methods.

The available methods of clinical biochemical analysis were modified so that the numerous determinations could be carried out on the smallest convenient specimen of blood. As will be seen, it was possible to carry out on six cubic centimetres of blood all the analyses with the exception of the determination of the blood volume.

The method of Hagedorn and Jensen was used for the glucose determination. The determination of urea content was carried out on 1·0 cubic centimetre of blood by the aeration method originally described by Van Slyke, which had previously been found to give accurate results (Bick[12]). The following modification of the existing Volhard method was used for the chloride determination:

Standard silver nitrate solution (two cubic centimetres) was added to two cubic centimetres of a protein-free filtrate prepared from 0·4 cubic centimetre of blood or 0·3 cubic centimetre of plasma by the method of Folin and Wu, and the excess silver nitrate was titrated with ammonium thiocyanate solution.

The alkali reserve was determined by the manometric method of Van Slyke, one cubic centimetre of plasma being used. Sahli's method was used for the hæmoglobin determination, 14 grammes of hæmoglobin representing 100%. The cell volume percentage was obtained by centrifuging 0·3 cubic centimetre of blood in hæmatocrit tubes for fifteen minutes. This at least provides comparable results. The serum calcium content was determined by a modification of the method described by Siwe.[13] The calcium was precipitated from 0·2 cubic centimetre of serum, and 0·0025N sodium thiosulphate solution was used for the final titration instead of the 0·01N sodium thiosulphate solution recommended. The plasma protein content was determined gravimetrically as follows:

To 0·1 cubic centimetre of plasma diluted with 0·6 cubic centimetre of water in small weighed tubes was added 0·1 cubic centimetre of $^{M}/_{15}$ sodium dihydrogen phosphate solution and a drop of amyl alcohol. After the mixture had been heated in a boiling water bath for thirty minutes, 1% acetic acid was added until the solution was at pH 6·4, when a flocculent precipitate was obtained. The heating was continued for a further fifteen minutes. The precipitate was then washed till free from phosphate ions by centrifuging with hot distilled water, finally with alcohol, and dried to constant weight.

The determination of the blood volume was carried out by means of the technique described by Bennett and his co-workers[14] for the collection of the blood and the preparation of the Congo red solution. Syringes calibrated to deliver were used for the injection of the dye. The colour comparison was made in a Huffner visible spectrophotometer as follows:

To 0·5 cubic centimetre of plasma was added 1·0 cubic centimetre of freshly distilled acetone, and the precipitate was removed by centrifugation. A solution of Congo red, of the same concentration as that injected, and diluted 500 times, was used as the standard and similarly treated.

Determinations of hæmoglobin in the presence of Congo red could also be carried out with this instrument, which had been previously calibrated at the optimum wavelengths against hæmoglobin and Congo red solutions of known concentrations.

The urinary calcium content was estimated by the method of Shohl and Pedley,[15] and the urinary glucose, urea and chloride contents were estimated by the methods used for the blood analyses after dilution of the urine (1 in 10) if necessary.

Case Records.

CASE I.—R.R., a male patient, aged forty-five years, was admitted to hospital on May 2, 1938, and died on June 17 (see Figure I). He had had flatulent dyspepsia for eight years, and a small hæmatemesis four years previously. On the day before his admission to hospital he had vomited 600 cubic centimetres of blood.

On examination he was very pale, apprehensive and slightly drowsy; his tongue was coated, but moist; slight epigastric tenderness was present, but no mass was palpable. The systolic blood pressure was 92 millimetres of mercury, the pulse rate was 105 per minute, and the hæmoglobin value was 39%; the alkali reserve was 58%, the plasma protein content was 6·2%, the blood urea level was 90 milligrammes *per centum*, and the blood sodium chloride content was 440 milligrammes *per centum*. The patient was given glucose saline solution by mouth and then the ulcer diet.

On May 3 the patient had a further hæmorrhage. The systolic blood pressure was 88 millimetres of mercury and the pulse rate was 90 per minute; the hæmoglobin value was 33%, the blood urea level 72 milligrammes *per centum* and the alkali reserve 64%; but the blood chloride content had risen to 540 milligrammes *per centum*. Little sodium chloride was excreted in the urine. A transfusion of 1,500 cubic centimetres of blood was given in four hours. The hæmoglobin value rose to 54%. There was no decrease in the blood urea level, but the patient felt much better and his colour was improved; 27·2 grammes of urea were excreted in the urine in twenty-four hours.

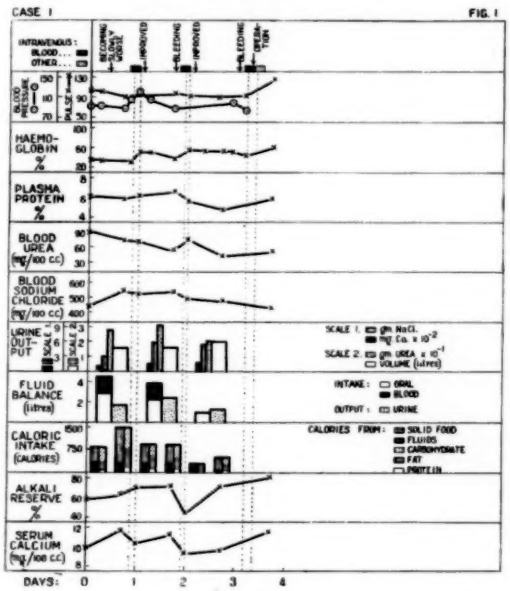

FIGURE I.

Showing the fall in blood pressure, hæmoglobin value, alkali reserve and plasma protein and chloride content, and the rise in the blood urea level occurring as a result of hæmorrhage. The effect of blood transfusion on these changes is shown.

On May 4 the patient was not so well. He was pale, and the hæmoglobin value was 39%. The plasma protein content was 6·7%, the alkali reserve 72% and the blood urea level 54 milligrammes *per centum*. A further transfusion of 1,500 cubic centimetres of blood was given in two and a quarter hours. After the transfusion the hæmoglobin value rose to 53%; but a fall occurred in the alkali reserve to 45%, in the plasma protein content to 5·6%, in the serum calcium content to 9·4 milligrammes *per centum*, and in the blood chloride content to 490 milligrammes *per centum*. The output of urine was still good, the 24-hour excretion of urea being 31·4 grammes; the excretion of chloride in the urine had also increased to 0·23%.

On May 5 the hæmoglobin value was 51% and the plasma protein content 4·8%. The patient became paler during the day and later vomited 180 cubic centimetres of blood. After the four severe hæmorrhages had been relieved by a transfusion of blood, it was decided to operate, and Dr. Syme found two chronic "kissing" ulcers in the first part of the duodenum. These were oversewn and a posterior gastro-enterostomy was performed. For the first twelve hours after operation the patient was very well indeed; but then he entered upon a stormy post-operative period, with gastric dilatation, pyrexia, pneumonia and empyema at the base of the right lung. Death occurred forty days after operation.

Death was due to an intraperitoneal leak, probably resulting from impaired vitality of the cells at the site of the anastomosis. The plasma protein level was low at the time of operation, and was only slightly improved by transfusion. The elevation of the plasma chloride content by the addition of sodium chloride to the food and drinks was most effective in the early stages of this case; but as the patient's condition became worse, the intake of sodium chloride by mouth could not be maintained. It is interesting to note the retention of sodium chloride in the tissues (see Figure V). A drop in the serum calcium level was found after each transfusion.

CASE II.—J.G., a male patient, aged twenty-two years, was admitted to hospital on October 27, 1938, and discharged on November 30, 1938. There was nothing of note in his past history. He had had a hæmatemesis twenty-four hours before his admission to hospital; the pulse rate was 105 per minute and the hæmoglobin value was 70%. He was given citrated milk, alkali, belladonna and morphine. His pallor very slowly increased, and he became thirsty, restless and drowsy, till he collapsed on November 2. On November 3 his systolic blood pressure was 112 millimetres of mercury and his pulse rate was 96 per minute; the hæmoglobin value was 39%, the alkali reserve 50%, the blood urea level 28 milligrammes *per centum*, the plasma sodium chloride content 460 milligrammes *per centum* and the serum calcium content 10·2 milligrammes *per centum*. The patient was given a transfusion of 1,200 cubic centimetres of blood in two hours, with great benefit. After the transfusion the hæmoglobin value was 55%, the alkali reserve 45%, the plasma protein content 6·9%, the blood urea content 27 milligrammes *per centum*, and the serum calcium content 9·6 milligrammes *per centum*.

On November 4 the patient's condition was improving; he was taking milk, eggs and larger volumes of fluid. The hæmoglobin value was 54%, the alkali reserve 52%, the plasma protein content 6·7%, the blood urea content 19 milligrammes *per centum*, and the plasma sodium chloride content 490 milligrammes *per centum*. On November 5 his condition was still improving; 15·6 grammes of urea had been excreted in the urine in the past twenty-four hours. The plasma chloride content was still low, and very little sodium chloride was excreted in the urine.

On November 6 the diet was increased to include semi-solid foods. On November 7 the hæmoglobin value was 56%, the alkali reserve 63%, and the plasma protein content 6·9%; the blood urea level was 29 milligrammes *per centum*, the plasma chloride content 520 milligrammes *per centum*, and the serum calcium content 10·6 milligrammes *per centum*.

The patient made an uneventful recovery. On November 24 the hæmoglobin value was 66%, the plasma protein level had risen to 7·1%, and the plasma chloride content to 560 milligrammes *per centum*. An X-ray examination was inconclusive, but suggested duodenal ulcer.

This young man suffered from a moderately slow but extensive hæmorrhage, which was soon relieved by blood transfusion. The initial biochemical analysis revealed a deficiency of sodium chloride, which was only slowly relieved while he was receiving a diet containing no added sodium chloride. A negligible amount of sodium chloride was excreted in the urine. The blood urea level did not rise, in spite of a poor excretion of urine containing a low concentration of urea. There was slight acidosis, and the serum calcium level fell during each transfusion.

CASE III.—W.Y., a male patient, aged sixty-two years, was admitted to hospital on March 17, 1939, and discharged on April 27 (Figure II). In 1928 he had had a perforated prepyloric ulcer; the ulcer was oversewn. In 1930 he suffered from severe indigestion; at operation a duodenal ulcer was found and posterior gastro-enterostomy was performed. In 1934 he had melena, which responded to treatment with alkali and rest. He had had vague dyspepsia and increasing weakness for three months prior to his admission to hospital, but had lost no weight. He had a severe hæmatemesis on the day before his admission to hospital.

On examination he was thin, pale and drowsy; his tongue was dry and brown, his systolic blood pressure was 90 millimetres of mercury, and his pulse rate was 100 per minute. The hæmoglobin value was 48%, the alkali reserve 44%, the plasma protein content 4·6%, the blood urea level 164 milligrammes *per centum*, the plasma sodium chloride content 460 milligrammes *per centum*, and the serum calcium content 10·6 milligrammes *per centum*. The bases of the lungs were congested, the abdomen was full, and epigastric tenderness was present.

The patient was given glucose saline solution by mouth for twelve hours; he would not take the ulcer diet. Later, on March 17, he began to vomit dark fluid and became drowsy; the vomiting persisted, and the alkali reserve fell to 37% and the plasma sodium chloride level from 510 to 470 milligrammes *per centum*; the blood urea level rose to 215 milligrammes *per centum*. He was given 1,200 cubic centimetres of blood and 2,400 cubic centimetres of Ringer's solution containing glucose. After these infusions the hæmoglobin value was 60%, the alkali reserve was 45%, and the plasma sodium chloride content was 550 milligrammes *per centum*. During these twenty-four hours the volume of urine excreted was very small—300 cubic centimetres—and the average urea concentration was 1·6%. No sodium chloride was excreted in the urine.

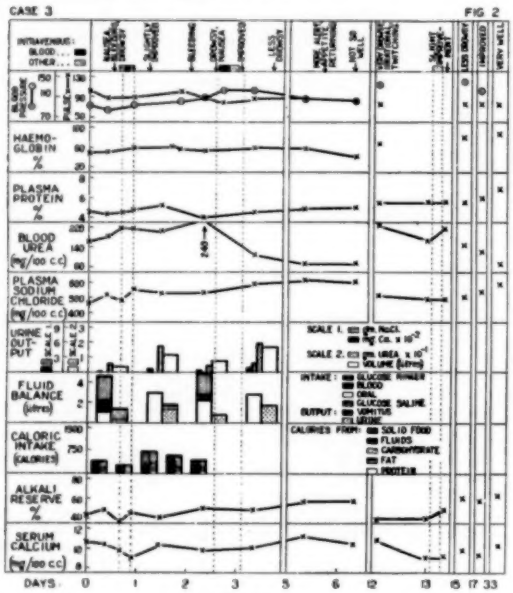

FIGURE II.

Showing biochemical changes associated with repeated hæmorrhage. There is a fall in the blood pressure, hæmoglobin value, plasma protein content, chloride content and alkali reserve. The abnormal elevation of the blood urea level to 240 milligrammes *per centum* is notable. The diminished excretion of urine, the poor caloric intake, the effects of the intravenous infusion of blood and saline solution on the clinical condition of the patient and the observed biochemical changes are also shown.

On March 18 he had a further hæmorrhage; the hæmoglobin value was 56%. He was drowsy, restless and nauseated; a larger volume of urine was excreted. On March 19 he had persistent hiccup and was refusing fluids; the hæmoglobin value was 53%, the alkali reserve 50%, the plasma protein content 4·0%, and the blood urea level 240 milligrammes *per centum*. The patient was *in extremis*. An infusion of 750 cubic centimetres of blood and two litres of glucose in saline solution was given. On March 20 improvement had followed the transfusion of blood and infusion of saline solution, and the blood urea level slowly fell to 110 milligrammes *per centum*; sodium chloride began to be excreted in the urine. On March 22 the patient's condition was still improving. The hæmoglobin value was 58%, the alkali reserve 56%, the plasma protein content 4·8%, the blood urea level 75 milligrammes *per centum*, the plasma sodium chloride content 610 milligrammes *per centum* and the serum calcium content 11·2 milligrammes *per centum*; the patient began to enjoy semi-solid food.

On March 24 he became drowsy and paler, and later he collapsed; he vomited 930 cubic centimetres of blood. The patient was *in extremis* again; the hæmoglobin value was 40% and the blood urea level 150 milligrammes *per centum*. He was given a massive intravenous infusion of 2,500 cubic centimetres of blood and 1,500 cubic centimetres of glucose and saline solution in eighteen hours. He revived slowly, but he was still desperately ill.

On March 26 twitching in the limbs and delirium were present; he was taking fluids well by mouth. A cough developed and increased pulmonary congestion was present. On March 28 the hæmoglobin value was 56%, the alkali reserve 34%, the plasma protein content 4·9%, the blood urea level 175 milligrammes *per centum*, the plasma sodium chloride content 550 milligrammes *per centum*, and the serum calcium content 10·9 milligrammes *per centum*. The patient was becoming more drowsy and irrational, and more twitching was present. Slight improvement followed an infusion of 600 cubic centimetres of blood and 750 cubic centimetres of glucose in saline solution. Calcium gluconate was given intravenously, without reduction of the twitching.

On March 29 the hæmoglobin value was 68%, the alkali reserve 38%, and the blood urea level 230 milligrammes *per centum*. The patient was taking fluids well.

On March 30 he was *in extremis* for the third time; he was drowsy, slightly cyanosed and suffering from air hunger. The alkali reserve was 40%, the blood urea level 163 milligrammes *per centum*, and the plasma sodium chloride content 480 milligrammes *per centum*. He was given one litre of Hartmann's 1/10 molar sodium lactate solution. His condition appeared to be improved; the alkali reserve was 48%.

On April 1 his condition was still improving; he was less drowsy and the twitching had ceased. The hæmoglobin value was 80%, the alkali reserve 60%, the plasma protein content 5·6%, the blood urea level 144 milligrammes *per centum*, the plasma sodium chloride content 500 milligrammes *per centum*, and the serum calcium content 9·9 milligrammes *per centum*. This improvement was maintained till his discharge from hospital on April 19; on that date the hæmoglobin value was 88%, the alkali reserve 64%, the plasma protein content 6·9%, the blood urea level 69 milligrammes *per centum*, the plasma sodium chloride content 580 milligrammes *per centum*, and the serum calcium content 10·2 milligrammes *per centum*. An X-ray examination revealed no abnormality; the stoma was functioning normally. A diagnosis was made of anastomotic ulcer (not proved), bronchitis and probable chronic nephritis.

This patient, on his admission to hospital, had a serious depletion of plasma protein and sodium chloride content, and a moderately severe degree of acidosis, despite the liberal administration of fluids. This acidosis persisted until the intravenous infusion of Hartmann's solution was given. The level of the plasma chloride was raised by the infusion of saline solution. The striking retention of sodium chloride in the tissues is shown in Figure V. The serious depletion of plasma protein content is not often found in these cases. There was no increase in the plasma protein level till the patient was able to take a liberal diet. It is interesting to note the refusal of almost all food and fluids in the early stages. It is possible that this patient had some preexisting renal damage, as it is unusual to find such high levels of blood urea (maximum reading, 240 milligrammes *per centum*). Moreover, there was poor urea concentration, even during convalescence. However, the urine contained no casts and never more than a trace of albumin. The dramatic effect of intravenous therapy is demonstrated, and the ultimate recovery of this patient in spite of his grave clinical condition and very high blood urea level astounded all concerned in his welfare. When seen nine months later, he said that he had enjoyed excellent health since his discharge from hospital.

CASE IV.—E.B., a male patient, aged seventy years, was admitted to hospital on August 26, 1939, and discharged on September 8 (see Tables IA and IB and Figure III). He had had mild flatulent dyspepsia for many years, and had lost one stone in weight during the past year. He had had hæmatemesis and melena two days prior to his admission to hospital, and had been allowed practically no fluid by mouth. The rectal administration of saline solution had been tolerated only for one day.

On examination he was pale, drowsy, thirsty and restless. His tongue was moderately dry and his abdomen full. The systolic blood pressure was 95 millimetres of mercury, the pulse rate 99 per minute and the hæmoglobin value 45%; the alkali reserve was 50%, the plasma protein level 6·1%, the blood urea level 56 milligrammes *per centum*, the plasma sodium chloride content 540 milligrammes *per centum*, and the serum calcium content 11·2 milligrammes *per centum*. The patient was given an ulcer diet.

On August 27 slight improvement was noticed; he was still thirsty. His systolic blood pressure was 120 millimetres of mercury; the plasma volume was 2·76 litres, the blood volume was 3·73 litres, the blood urea level was 54

TABLE IA.[1]

Class IV: E.B., a male patient, aged seventy years. Diagnosis: probable duodenal ulcer, hæmatemesis. Result: recovery.

Day of Observation.	Time.	Clinical Condition.	Blood.												Urine.				
			Pulse Rate per Minute.	Systolic Blood Pressure. (Millimetres of Mercury.)	Hæmoglobin Value. (Percentage.)	Cell Volume. (Percentage.)	Plasma Volume. (Cubic Centimetres.)	Blood Volume. (Cubic Centimetres.)	Alkali Reserve. (Percentage.)	Plasma Protein Content. (Percentage.)	Urea. (Milligrammes per 100 Cubic Centimetres.)	Plasma Sodium Chloride. (Milligrammes per 100 Cubic Centimetres.)	Glucose. (Milligrammes per 100 Cubic Centimetres.)	Serum Calcium. (Milligrammes per 100 Cubic Centimetres.)	Volume (Cubic Centimetres.)	Urea (Grammes.)	Sodium Chloride. (Gramme.)	Glucose. (Gramme.)	Calcium. (Milligrammes.)
1	p.m. 8.0	Bleeding two days. Drowsy, restless, thirsty, tongue moderately dry. On restricted food and fluid by mouth for two days, has had rectal administration of saline solution. Probable hæmorrhage six hours previously..																	
	a.m. 5.30		99	95	45	27	—	—	50	6·1	56	540	110	11·2	240 360	2·4 8·6	Nil Nil	0·6 0·5	31 43
	11.0	Condition slightly improved. Thirsty, heavy breath. Salt given by mouth in capsules ..																	
	p.m. 2.0	Still improving. Receiving citrated milk..		120		26	2760	3730	54	6·0	58	450	80	9·4	390	3·7	0·3	0·7	59
2	p.m. 8.30	Occasional slight abdominal pain ..													150	1·4	Nil	0·2	19
	a.m. 8.0	Condition improving. No sign of fresh bleeding..													390	3·1	0·4	0·8	57
	9.30	Less drowsy. Colour																	
	p.m. 2.30	slightly better .. Comfortable.		125	45	26			62	6·3	24	500	150	10·7	240	3·8	0·3	0·4	31
3	p.m. 8.0														300	4·4	0·1	0·4	33
	a.m. 8.0	Occasional slight abdominal pain, otherwise well, much more																	
	md. 12	alert		125	47		—	—	60	6·7	24	530	110	10·6	570 360	4·8 3·3	0·5 0·3	0·5 0·3	58 28
4	p.m. 9.0														360	2·8	0·4	0·7	48
	a.m. 8.0	Condition improving													360	4·7	0·3	0·9	28
	9.30	Sodium chloride capsules discontinued, diet increased	68		50	30	3060	4370	62	6·7	22	570	130	9·4					
13	a.m. 9.30	Excellent. Sitting out of bed.[2]																	
	p.m. 12.30			112	67	35	3270	5030	68	7·0	16	560	—	10·7	300	3·8	0·2	—	39
	2.30														180	2·3	0·2	—	18

[1] This table shows the biochemical changes associated with severe hæmorrhage. Improvement in the patient's condition following rest and adequate fluids and food by mouth is found to be associated with the correction of these abnormal changes. The slow restoration of the blood volume is to be noted. In columns under "Urine" the quantities in any one line represent the amounts passed during the time between the last and the given specimen.

[2] X-ray examination one month later revealed no lesion. The patient was in excellent health four months later.

milligrammes *per centum*, and the plasma chloride content had decreased to 450 milligrammes *per centum*. He was given a liberal amount of fluids and capsules of sodium chloride by mouth.

On August 28 his condition was improving; the hæmoglobin value was 45%, the alkali reserve was 62%, the blood urea level had fallen to 24 milligrammes *per centum*, and the plasma chloride level had risen to 500 milligrammes *per centum*. The amount of urea excreted in the urine in the past twenty-four hours was 13·7 grammes. On August 29 his condition was still improving. The systolic blood pressure was 125 millimetres of mercury, the hæmoglobin value was 47%, and the blood urea level was 24 milligrammes *per centum*; the plasma protein level had risen to 6·7% and the plasma chloride content to 530 milligrammes *per centum*. There was also an increase in the volume of urine, and 11·3 grammes of urea had been excreted in the past twenty-four hours; the amount of chloride excreted had increased very little.

On August 30 the patient was taking eggs, fish, vegetable *purée*, citrated milk, "Marmite", orange juice and iron tablets. The administration of sodium chloride capsules was discontinued. The hæmoglobin value was 50%, the plasma volume was 3·06 litres and the blood volume was 4·37 litres; the plasma chloride content was 570 milligrammes *per centum* and the blood urea level was 22 milligrammes *per centum*. On August 31 his condition was much improved, and he was sitting out of bed. On September 8 his condition was excellent; the systolic blood pressure was 112 millimetres of mercury, the hæmoglobin value 67%, the plasma volume 3·27 litres, the blood volume 5·03 litres, the alkali reserve 68%, the plasma protein content 7·0%, the blood urea level 16 milligrammes *per centum*, and the plasma sodium chloride content 560 milligrammes *per centum*. An X-ray examination revealed no abnormality. When the patient was seen four months later he was still enjoying excellent health.

This case illustrates the decrease in the blood urea level following the oral administration of adequate amounts of fluid. The appetite was poor until a definite improvement occurred in the patient's general condition. On the first day he consumed 380 Calories and on the second day 686. The depletion of sodium chloride was

TABLE IB.[1]

Case IV: Oral Intake.

Day of Observation.	Time.	Carbohydrate. (Grammes.)	Protein. (Grammes.)	Fat. (Grammes.)	Calories from Fluid Food.	Total Calories.	Volume of Water *plus* Water Content of Food. (Cubic Centimetres.)	Calcium. (Gramme.)	Sodium Chloride. (Grammes.)
1	7 p.m. to 1 a.m. ..	6	4	4	76	76	360	0·14	0·21
	1 a.m. to 7 a.m. ..	3	2	2	38	38	300	0·07	0·11
	7 a.m. to 1 p.m. ..	12	8	8	152	152	660	0·28	0·42
	1 p.m. to 7 p.m. ..	9	6	6	114	114	670	0·21	1·41
2	7 p.m. to 1 a.m. ..	0	0	0	0	0	150	0	0
	1 a.m. to 7 a.m. ..	3	2	2	38	38	90	0·07	0·11
	7 a.m. to 1 p.m. ..	36	8	9	76	257	780	0·33	1·38
	1 p.m. to 7 p.m. ..	35	11	23	198	391	630	0·26	1·54
3	7 p.m. to 1 a.m. ..	6	4	5	85	85	450	0·14	0·21
	1 a.m. to 7 a.m. ..	0	0	0	0	0	150	0·0	0·0
	7 a.m. to 1 p.m. ..	42	12	15	154	351	570	0·29	1·58
	1 p.m. to 7 p.m. ..	56	18	31	0	575	880	0·34	0·99
4	7 p.m. to 1 a.m. ..	25	12	14	274	274	630	0·26	1·58
	1 a.m. to 7 a.m. ..	—	—	—	—	—	—	—	1·10
	7 a.m. to 1 p.m. ..	—	—	—	—	—	—	—	—
	1 p.m. to 7 p.m. ..	—	—	—	—	—	—	—	—

[1] This table shows the low caloric intake by mouth found in these cases and its subsequent increase.

relieved by the addition of sodium chloride capsules to the diet. The plasma protein level was raised when the amount of protein in the diet was increased. It is interesting to note the serious depletion of the blood volume and the slow increase in both cell and plasma volume. Three days after the initial determination there was an increase of 300 cubic centimetres in the plasma volume, accompanied by a 4% increase in the cell volume.

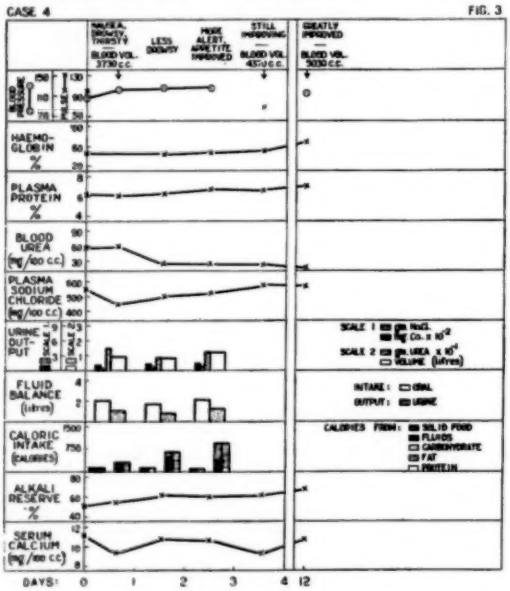

FIGURE III.

Showing the depletion of blood volume, the fall in hæmoglobin value, plasma protein, chloride content and alkali reserve, and the rise in blood urea level, occurring as results of hæmorrhage. The influence of rest and adequate fluid intake on these changes, together with the low caloric intake, is also shown.

This elevated the blood volume by 640 cubic centimetres. After a further nine days the blood volume was increased by 660 cubic centimetres; the increase in plasma volume was 210 cubic centimetres. This case provides a good example of what appears to be the most rational method of treating a moderately severe hæmorrhage from the stomach or duodenum.

CASE V.—D.H., a male patient, aged thirty years, was admitted to hospital on November 7, 1938, and discharged on November 30. For the past year he had consumed large quantities of alcohol. He had had recurrent attacks of vomiting of blood, with increasing pallor, for the three days before his admission to hospital. On examination he was pale, restless and apprehensive, but not collapsed. On November 8 he was still vomiting blood and was becoming drowsy and collapsed. His pulse rate was 120 per minute and the pulse was of poor volume; the hæmoglobin value was 50%. He was given a transfusion of 750 cubic centimetres of blood in twenty-three minutes, with great benefit: the hæmoglobin value rose to 65%; the alkali reserve was 48%, the plasma protein content 6·1%, the blood urea level 65 milligrammes *per centum*, the plasma sodium chloride content 470 milligrammes *per centum*, and the serum calcium content 10·4 milligrammes *per centum*.

On November 9 the improvement was maintained; the patient was a better colour, more confident and less restless; he was having milk, "Benger's Food", orange juice and alkali. The alkali reserve was 60%, the plasma protein content 6·8%, the blood urea level had decreased to 27 milligrammes *per centum*, the plasma sodium chloride content had increased to 540 milligrammes *per centum*, and the serum calcium content was 11·5 milligrammes *per centum*. On November 10 his condition was still improving and his diet was increased.

On November 24 his condition was excellent; the systolic blood pressure was 120 millimetres of mercury; the hæmoglobin value was 80%, the alkali reserve 72%, the plasma protein content 8·3%, the blood urea level 31 milligrammes *per centum*, the plasma sodium chloride content 570 milligrammes *per centum*, and the serum calcium content 10·5 milligrammes *per centum*. An X-ray examination revealed no abnormality; a diagnosis of probable alcoholic gastritis was made.

Blood transfusion and the liberal administration of fluids by mouth were followed by rapid improvement in the clinical condition. This was accompanied by a decrease in the blood urea level and an increase in the alkali reserve and plasma chloride content. A vitamin deficiency may have been present. No record was made of the excretion of urine.

CASE VI.—G.H., a male patient, aged forty-four years, was admitted to hospital on March 29, 1938, and discharged on April 2. He had had recurrent attacks of dyspepsia of duodenal type for ten years. Four days prior to his admission to hospital he vomited 600 cubic centimetres of blood and later passed tarry stools. On examination he was very pale and slightly restless; his tongue was moist, but coated, and his abdomen was normal. The systolic blood pressure was 106 millimetres of mercury and the pulse rate 156 per minute. The hæmoglobin value was 48%, the alkali reserve 61%, the plasma protein content 6·9%, the blood urea level 43 milligrammes *per centum*, the blood sodium chloride content 320 milligrammes *per centum*, and the serum calcium content 11·8 milligrammes *per centum*. The patient was given a modified Meulengracht diet, with feedings every two hours.

On March 30 he had a further hæmatemesis and vomited 140 cubic centimetres of blood; the hæmoglobin value fell to 33%, although the plasma chloride content increased to

430 milligrammes *per centum* and the blood urea level fell to 38 milligrammes *per centum*. A massive transfusion of 1,800 cubic centimetres of blood was given in four hours. After the transfusion the hæmoglobin value was 53% and the plasma sodium chloride content was 430 milligrammes *per centum*; the serum calcium content fell to 10·2 milligrammes *per centum* and the blood urea level rose to 45 milligrammes *per centum*. This increase in the blood urea level following transfusion is unusual.

On March 31 the improvement was maintained: the systolic blood pressure was 116 millimetres of mercury, the pulse rate was 92 per minute and the hæmoglobin value was 60%; the plasma protein content was 7·0%, the blood urea level 39 milligrammes *per centum*, the blood chloride content 480 milligrammes *per centum*, and the serum calcium content 10·2 milligrammes *per centum*. The volume of urine excreted was large; 29·5 grammes of urea had been excreted in the past twenty-four hours; the average concentration of chloride in the urine was 0·34%.

On April 21 the hæmoglobin value was 86%; the alkali reserve was 70%, the plasma protein content 7·7%, the blood urea level 21 milligrammes *per centum*, the blood sodium chloride content 520 milligrammes *per centum*, and the serum calcium content 10·2 milligrammes *per centum*. Examination after a test meal revealed hyperchlorhydria and an X-ray examination revealed a duodenal ulcer. A gastroscopic examination revealed no abnormality. A diagnosis of duodenal ulcer was made.

The low blood chloride content was associated with a diminished chloride content in the urine. The plasma protein content was slightly depleted and the blood urea level was elevated. A blood transfusion was given, with improvement in the patient's clinical condition.

CASE VII.—A.L., a male patient, aged twenty-two years, was admitted to hospital on April 20, 1939, and died on April 30 (see Table II). He had had indigestion for four years and hæmatemesis on two previous occasions. In 1937 an X-ray examination had revealed a duodenal ulcer. He had had severe melena and had collapsed two days prior to his admission to hospital; he had also suffered from thirst and drowsiness. On his admission he was drowsy, restless, pale and very thirsty; the systolic blood pressure was 110 millimetres of mercury, the pulse rate was 108 per minute and the hæmoglobin value was 52%.

Four hours later the hæmoglobin value was 50%, the alkali reserve 38%, the plasma protein content 4·7%, the blood urea level 84 milligrammes *per centum*, the plasma

TABLE II.[1]

Case VII: A.L., a male patient, aged twenty-two years. Diagnosis: Duodenal ulcer, hæmatemesis. Result: Death.

Day of Observation.	Time.	Clinical Condition.	Blood.									Urine.				
			Pulse Rate per Minute.	Systolic Blood Pressure. (Millimetres of Mercury.)	Hæmoglobin Value. (Percentage.)	Cell Volume. (Percentage.)	Alkali Reserve. (Percentage.)	Plasma Protein. (Percentage.)	Urea Level. (Milligrammes per 100 Cubic Centimetres.)	Plasma Sodium Chloride. (Milligrammes per 100 Cubic Centimetres.)	Glucose. (Milligrammes per 100 Cubic Centimetres.)	Serum Calcium. (Milligrammes per 100 Cubic Centimetres.)	Volume. (Cubic Centimetres.)	Urea. (Grammes.)	Sodium Chloride. (Grammes.)	Glucose. (Gramme.)	Calcium. (Milligrammes.)
1	a.m. 11.0	Bleeding three days. Drowsy, restless, dry tongue, very thirsty	108	110	52								210	5·3	0·3	0·5	32
	p.m. 3.0	Transfusion 900 cubic centimetres in two hours			50	24	38	4·7	84	480	150	9·0					
	5.0	End of transfusion. Condition improved. Infusion of 3% sodium chloride solution commenced	94		61	30	41	5·5	73	520	120	9·6					
2	a.m. 8.0	Received two litres of 3% sodium chloride solution intravenously. Taking semi-solid food. Restless	104	110									540	11·3	0·7	0·9	58
	9.0				50	20	48	5·1	59	650	110	8·9					
	10.0	Still restless											570	13·5	5·5	0·4	50
	p.m. 5.0	Pale, collapsed, sweating; hæmatemesis. *In extremis.* Blood transfusion commenced..	128		28	14	40	3·7	49	470	90	9·6					
	10.0	Condition improving											300	1·5	2·3	0·4	20
3	a.m 1.0												330	2·4	2·3	0·6	26
	5.0	Condition improved. Less restless. Colour better											540	6·2	3·9	0·8	22
	9.0	End of infusion. Has received 2·5 litres of blood and 300 cubic centimetres of 5% glucose in normal saline solution ..	120	110	56	24	43	4·5	39	480	110	10·1					
	p.m. 12.30												360	5·4	2·2	1·1	26
	3.0	Just after operation, lasting two hours, and infusion of 5% glucose in normal saline solution, one litre, and blood, 150 cubic centimetres	98		52	25	44	4·8	29	550	150	7·7					
	9.0												360	2·4	2·1	0·9	14
4	a.m. 1.0		100										390	5·9	2·5	1·2	28
	4.0												300	2·6	2·6	0·7	26
	9.0[2]	Condition improving. No evidence of further hæmorrhage. In 18 hours following operation has received blood, 600 cubic centimetres, 5% glucose in normal saline solution, 750 cubic centimetres, and sodium lactate solution, 500 cubic centimetres	90	130	62	30	44	4·9	27	550	130	8·5					

[1] This table shows the biochemical changes observed as a result of repeated hæmorrhage. The effect of transfusion and the intravenous infusion of saline solution on these changes and on the clinical condition of the patient are shown. The pronounced degree of acidosis and the severe depletion of the plasma protein is of interest in this case. The caloric intake by mouth was negligible. In columns under "Urine", the quantities in any one line represent the amounts passed during the time between the last and the given specimen.

[2] At this stage the patient slowly developed the manifestations of intestinal obstruction and subsequently died.

sodium chloride content 480 milligrammes *per centum*, and the serum calcium content 9·0 milligrammes *per centum*. A transfusion of 900 cubic centimetres of blood was given in two hours. After the transfusion the hæmoglobin value was 61%, the alkali reserve 41%, the plasma protein content 5·5%, the blood urea level 73 milligrammes *per centum*, and the plasma sodium chloride content 520 milligrammes *per centum*. His condition appeared to be improved and he was less restless after the transfusion, which was immediately followed by the infusion of two litres of 3% sodium chloride solution in fifteen hours. He appeared to be more alert, and was taking semi-solid food.

On April 21 the systolic blood pressure was 110 millimetres of mercury, the pulse rate was 104 per minute and the hæmoglobin value was 50%; the alkali reserve was 48%, the plasma protein content 5·1%, the plasma sodium chloride content 650 milligrammes *per centum*, and the serum calcium content 8·9 milligrammes *per centum*. In the past twenty-four hours the blood urea level had fallen from 73 to 59 milligrammes *per centum*, and 30·1 grammes of urea had been excreted in the urine, the urea concentration being 2·3%. The excretion of sodium chloride in the urine was also high—0·56%. Eight hours later the patient had a severe hæmatemesis and death appeared imminent; the pulse rate was 128 per minute, and the hæmoglobin value had fallen to 28%, the alkali reserve to 40%, the blood urea level to 49 milligrammes *per centum*, the plasma protein content to 3·7%, and the plasma sodium chloride content to 470 milligrammes *per centum*. He was given 2·5 litres of blood and 300 cubic centimetres of 5% glucose in normal saline solution in sixteen hours. At the end of the infusion the systolic blood pressure was 110 millimetres of mercury, the pulse rate was 120 per minute and the hæmoglobin value was 56%; the alkali reserve was 43%, the plasma protein content 4·5%, the blood urea level 39 milligrammes *per centum*, the plasma sodium chloride content 480 milligrammes *per centum*, and the serum calcium content 10·1 milligrammes *per centum*. Although the plasma chloride content was low, the amount of chloride excreted in the urine remained high. The volume of urine was still large (1,170 cubic centimetres); but only 10·1 grammes of urea had been excreted in the urine in the past twenty-four hours, the average concentration being 0·9%.

Operation was then performed and a chronic duodenal ulcer was found on the posterior surface of the duodenum. This was oversewn, the duodenum was occluded, and a gastro-enterostomy was performed. During the next twenty-four hours the patient was given a further infusion of blood, followed by 5% glucose in saline solution and Hartmann's sodium lactate solution. His condition was quite satisfactory.

On April 23 the systolic blood pressure was 130 millimetres of mercury, the pulse rate was 90 per minute and the hæmoglobin value was 62%, the alkali reserve was 44%, the plasma protein content 4·9%, the blood urea level 27 milligrammes *per centum*, the plasma sodium chloride content 550 milligrammes *per centum*, and the serum calcium content 8·5 milligrammes *per centum*. During the past twenty-four hours 16·3 grammes of urea had been excreted in the urine. Later on this day the patient began to vomit, but he suffered practically no abdominal pain. The vomiting persisted in spite of gastric aspiration and other measures aimed at the relief of the gastric dilatation and paralytic ileus. Slight jaundice also appeared later, and the patient died nine days after his admission to hospital.

On the day prior to his death he experienced a sudden severe mid-abdominal pain, which soon became generalized. A second laparotomy was performed, and it was found that a small loop of jejunum, just distal to the anastomosis, was bound down and obstructed by a fibrous adhesion. This adhesion formed part of the wall of a small intraperitoneal abscess. The jejunum had recently perforated immediately proximate to the obstruction. A diagnosis of duodenal ulcer and hæmorrhage was made. The operation revealed pyloric exclusion, intraperitoneal abscess, intestinal obstruction, perforated jejunum and general peritonitis.

This case illustrates the decrease in blood urea content that follows adequate fluid intake. Moreover, determination of the blood urea level immediately after a massive hæmorrhage revealed no rise. A liberal infusion of blood followed by glucose in saline solution was begun in less than an hour, and this was followed by a still further fall in the blood urea level. This suggests that it probably takes several hours for the blood urea level to rise after a massive hæmorrhage, and that the rise can be prevented or lessened by the prompt administration of fluid. The protein depletion was more serious in this case, and although the plasma protein level was raised by transfusion, this increase was not maintained. This serious depletion of protein is unusual. The acidosis was also severe, and did not respond to either glucose and saline solution or Hartmann's sodium lactate solution. The plasma chloride level was rapidly raised by the intravenous infusion of 3% sodium chloride solution, and chloride appeared in the urine in high concentration—0·56%. It is surprising to find that this excretion of chloride was maintained in spite of a subsequent fall in the plasma chloride content. The serum calcium level was low in this case, and possibly Ringer's solution could with advantage have been substituted for glucose and saline solution.

Chronic peptic ulcer in such a young man is unusual. The detrimental effects on the tissues of severe anæmia and the associated biochemical changes are strikingly demonstrated. Death was probably due to an intraperitoneal leak at the site of the anastomosis.

CASE VIII.—J.B., a male patient, aged sixty-seven years, was admitted to hospital on July 22, 1938, and discharged on August 18. He had had a severe hæmatemesis three years previously, but no dyspepsia. He had been well till twelve hours before his admission to hospital, when he vomited 600 cubic centimetres of blood and passed a tarry stool. On examination he was pale and slightly restless; his tongue was dry and brown and his abdomen was normal; the pulse rate was 99 per minute, the systolic blood pressure was 110 millimetres of mercury and the hæmoglobin value was 45%. He was given milk, egg flip and orange drinks, to which salt was added.

Two days later he became pale and restless; the hæmoglobin value was 47%, the systolic blood pressure was 106 millimetres of mercury, and the pulse rate 104 per minute; the alkali reserve was 47%, the plasma protein content 6·0%, the blood urea level 71 milligrammes *per centum*, the plasma sodium chloride content 590 milligrammes *per centum*, and the serum calcium content 10·2 milligrammes *per centum*. The patient was able to take the diet well.

On July 26 his condition had improved. The blood urea level was 77 milligrammes *per centum*, and 18·6 grammes of urea had been excreted in the urine in the past twenty-four hours. The hæmoglobin value was falling; at 7 p.m. it was 39%. The systolic blood pressure was 106 millimetres of mercury, the pulse rate was 78 per minute, and the volume of urine excreted was good; some chloride was excreted in the urine.

On July 27 the systolic blood pressure was 116 millimetres of mercury, the pulse rate was 86 per minute, and the hæmoglobin value was 39%. The plasma protein content had fallen slightly to 5·5% and the blood urea level had fallen to 51 milligrammes *per centum*. The serum calcium content was 9·9 milligrammes *per centum*. The excretion of sodium chloride in the urine was greater (0·28%), and 20·4 grammes of urea had been excreted in the past twenty-four hours. The patient's condition continued to improve with rest, diet and the administration of alkali. No transfusion was given, and the increase in hæmoglobin percentage was slow.

On July 29 the hæmoglobin value was 42%, the alkali reserve 56%, the plasma protein content 6·0%, the blood urea level 30 milligrammes *per centum*, the plasma sodium chloride content 580 milligrammes *per centum*, and the serum calcium content 11·0 milligrammes *per centum*. Examination after a test meal revealed hyperchlorhydria, and an X-ray examination, though inconclusive, was suggestive of duodenal ulcer.

This case illustrates the elevation of the blood urea level following hæmorrhage and its subsequent lowering following an adequate intake of fluid. On July 26 the blood urea level fell from 77 to 51 milligrammes *per centum*. During this period the patient took 2,820 cubic centimetres of fluid and food by mouth, and passed 1,400 cubic centimetres of urine containing 20·4 grammes of urea, an average urea concentration of 1·5%. A slight degree of acidosis and some depletion of the plasma protein content were present. There was no deficiency of sodium chloride content in the plasma, owing to the liberal administration of sodium chloride in the diet when the patient was first admitted to hospital. A blood transfusion was considered for this patient, but was withheld, owing to his satisfactory clinical appearance, the good pulse and blood pressure, and the falling blood urea level. This shows the satisfactory treatment of hæmatemesis with adequate diet, fluids and sodium chloride.

CASE IX.—T.K., a male patient, aged sixty-nine years, was admitted to hospital on March 14 and died four hours afterwards. He had had dyspepsia for one year. Flatulence

and occasional vomiting had been present for two weeks prior to his admission to hospital, and melena for three days.

On examination the patient was *in extremis*. Gross pallor was present; the pulse rate was 140 per minute and the systolic blood pressure was 98 millimetres of mercury, and falling; the hæmoglobin value was 21%, the alkali reserve 52%, the plasma protein content 5·2%, the blood urea level 130 milligrammes *per centum*, the sodium chloride content 490 milligrammes *per centum*, and the serum calcium content 10·9 milligrammes *per centum*. No transfusion of blood was given.

At autopsy a chronic duodenal ulcer with an eroded artery was found, together with renal fibrosis.

Immediate rapid transfusion of stored blood might have saved this patient. This was not available.

CASE X.—J.H., a male patient, aged sixty-one years, was admitted to hospital on April 23, 1938, and discharged on May 24. He had had dyspepsia occasionally for twelve years, and hæmatemesis ten years earlier. He had had epigastric pain for five days prior to his admission to hospital, followed four days later by hæmatemesis. On examination he was pale, but not restless; his tongue was moist and coated, and epigastric tenderness was present; the pulse rate was 77 per minute and the systolic blood pressure 82 millimetres of mercury; the hæmoglobin value was 68%, the alkali reserve 56%, the plasma protein content 7·2%, the blood urea level 61 milligrammes *per centum*, and the sodium chloride content 420 milligrammes *per centum*.

On April 27 his condition was greatly improved. The pulse rate was 67 per minute, the hæmoglobin value 80%, the alkali reserve 67%, the plasma protein content 6·7%, the blood urea level 21 milligrammes *per centum*, and the sodium chloride content 460 milligrammes *per centum*.

He made an uneventful recovery with rest, diet and the administration of alkaline powders. An X-ray examination and a gastroscopic examination revealed no abnormality. Examination after a test meal revealed no abnormality. A diagnosis of acute peptic ulcer was made, but not proved.

The low plasma chloride content and the absence of chloride in the urine are of interest. The blood urea level was elevated.

CASE XI.—H.A., a male patient, aged forty-one years, was admitted to hospital on January 8, 1938. There was nothing of note in his past history. He had had epigastric discomfort for two weeks, brought on by food and relieved by alkali, and a small hæmatemesis six hours prior to his admission to hospital.

On examination he appeared to be well. His liver was enlarged to one inch below the right costal margin. The hæmoglobin value was 90%, the pulse rate was 88 per minute, and the systolic blood pressure was 120 millimetres of mercury. The alkali reserve was 60%, the plasma protein content 7·0%, the blood urea level 32 milligrammes *per centum*, the plasma sodium chloride content 490 milligrammes *per centum*, and the serum calcium content 10·5 milligrammes *per centum*. Examination after a test meal revealed achlorhydria. An X-ray examination revealed no abnormality and the Wassermann test failed to produce a reaction. A diagnosis of gastritis was made, but not confirmed.

In this case slight hæmorrhage caused practically no abnormal biochemical changes.

CASE XII.—E.F., a male patient, aged thirty-one years, was admitted to hospital on November 14, 1938, and discharged on December 10. He had had flatulent dyspepsia for three months and hæmatemesis and melena for four days.

On examination mild pallor was noted, but there was no toxæmia. The condition responded to rest, diet and the administration of alkali. On November 14 the pulse rate was 108 per minute, the systolic blood pressure was 122 millimetres of mercury, and the hæmoglobin value was 72%. The plasma protein content was 5·8%, the blood urea level 49 milligrammes *per centum*, the plasma sodium chloride content 570 milligrammes *per centum*, and the serum calcium content 10·6 milligrammes *per centum*.

On November 18 the pulse rate was 76 per minute, the hæmoglobin value 76%, the plasma protein content 7·4%, the blood urea level 22 milligrammes *per centum*, the plasma sodium chloride content 610 milligrammes *per centum*, and the serum calcium content 10·6 milligrammes *per centum*. An X-ray examination revealed no lesion. Examination after a test meal revealed achlorhydria. A diagnosis of gastritis was made, but not confirmed.

The moderately low plasma protein content improved with suitable feeding of the patient. The achlorhydria is of interest.

CASE XIII.—A.B., a male patient, aged thirty-five years, was admitted to hospital on July 4, 1938, and discharged on July 25. He had had chronic dyspepsia for ten years. For five days prior to his admission to hospital he had had severe indigestion with occasional vomiting; streaks of blood were present in the vomitus.

On examination he was not pale; epigastric tenderness was present. The pulse rate was 64 per minute, the diastolic blood pressure 115 millimetres of mercury, and the hæmoglobin value 105%. The alkali reserve was 54%, the plasma protein content 7·9%, the blood urea level 37 milligrammes *per centum*, the sodium chloride content 530 milligrammes *per centum*, and the serum calcium content 9·9 milligrammes *per centum*.

An X-ray examination revealed a duodenal ulcer, and examination after a test meal revealed hyperchlorhydria. A diagnosis of duodenal ulcer was made.

When the hæmorrhage was slight there were no biochemical changes of note.

Discussion.

Blood Pressure.

The blood pressure falls during a massive hæmorrhage. After the hæmorrhage has ceased the blood pressure tends to rise again, the rate and extent of the rise depending on the available tissue fluid, the state of the cardiovascular system and the replacement of shed blood by transfusion. If the bleeding is slow the blood pressure may be maintained within normal limits. Even if given moderately rapidly (600 cubic centimetres per hour), a blood transfusion causes the blood pressure to rise, but the pressure is not forced to abnormal heights; on the contrary, it more frequently remains subnormal.

Hæmoglobin.

For many years physicians have considered that a determination of the hæmoglobin percentage is a valuable guide to the degree of exsanguination of the patient, and may indicate the onset of further bleeding. With the introduction of more accurate methods of determining the percentage of hæmoglobin, and with the realization that the pulse and blood pressure may not alter during a slow hæmorrhage, the hæmoglobin percentage has in recent years been considered to be even more informative than was previously thought. Only when the blood volume is fully restored is the hæmoglobin percentage a true index of circulatory hæmoglobin. After a hæmorrhage, when the blood volume is depleted, the hæmoglobin percentage is high in relation to the total amount of circulating hæmoglobin. Then, as the blood volume is restored by the tissue fluids, the hæmoglobin falls. This fall, which takes place after the hæmorrhage has ceased, may be falsely interpreted as indicating further bleeding (Case VIII). Bennett and his co-workers[4] found in a series of cases of gastric and duodenal hæmorrhage that in some the plasma volume recovered completely in a few hours, but in others not till six to twenty-four hours had elapsed. The plasma volume was not increased above normal to compensate for cell loss. Robertson,[6] however, states that the total blood volume is rapidly and fully restored to normal, implying that the plasma volume increases above normal.

In the only case of hæmatemesis in the present series in which the blood volume was studied (Case IV), the initial determination was carried out some twenty-four hours after the hæmorrhage had occurred, and showed that the blood volume was seriously depleted. The subsequent increase in both the plasma and cell volume was slow. The slow recovery rate of the plasma volume in this case was probably attributable to the inability to mobilize sufficient fluid from the tissue spaces owing to general dehydration. Moreover, the limited consumption of fluid by mouth would be inadequate to replace any previous excessive loss. The blood volume determinations carried out on a control subject under the same conditions over a period of ten days remained relatively constant (Table IIIA, Figure IV).

It appears, in the light of clinical and laboratory observations, that although the percentage of hæmoglobin does not give a completely accurate index of the extent of blood loss, it is sufficiently helpful to be of great value

TABLE IIIA.[1]
"Control B."

Day of Observation.	Time.	Blood.												Urine.				
		Pulse Rate per Minute.	Systolic Blood Pressure. (Millimetres of Mercury.)	Hæmoglobin Value. (Percentage.)	Cell Volume. (Percentage.)	Plasma Volume. (Cubic Centimetres.)	Blood Volume. (Cubic Centimetres.)	Alkali Reserve. (Percentage.)	Plasma Protein. (Percentage.)	Urea. (Milligrammes per 100 Cubic Centimetres.)	Plasma Sodium Chloride. (Milligrammes per 100 Cubic Centimetres.)	Glucose. (Milligrammes per 100 Cubic Centimetres.)	Serum Calcium. (Milligrammes per 100 Cubic Centimetres.)	Volume (Cubic Centimetres.)	Urea (Grammes.)	Sodium Chloride. (Gramme.)	Glucose. (Gramme.)	Calcium. (Milligrammes.)
1	9.0 a.m.				45	3310	6190	60	8·2	30	620	90	10·9					
	12.0 m.d.													240	3·9	0·7	0·4	60
	2.30 p.m.													300	4·1	1·5	1·0	77
	8.0 p.m.													240	3·9	0·7	0·4	60
	9.0 p.m.				44			62	8·1	19	580	70	10·6					
	11.0 p.m.													120	1·9	0·4	0·2	30
2	5.0 a.m.													240	2·6	1·2	0·2	79
	7.0 a.m.													270	1·5	0·6	0·4	72
	9.0 a.m.							65	8·2	16	590	110	11·2					
	11.0 a.m.													120	1·3	0·9	0·2	33
	1.30 p.m.													210	3·6	1·1	0·4	76
	5.0 p.m.	70	110	94										200	2·3	0·9	0·3	51
	9.0 p.m.				44			62	8·2	17	590		10·6					
	12.0 m.n.													110	2·0	0·5	0·2	39
3	7.0 a.m.													470	7·9	2·2	0·9	94
	8.0 a.m.													30	0·1	0·2	Nil	9
	9.0 a.m.				44			65	8·1	29	600	90	10·4					
	11.0 a.m.													350	1·5	1·4	0·6	102
	5.0 p.m.													540	6·5	3·3	1·0	140
	9.0 p.m.	70	110	95	45			63	8·2	21	580	90	10·7					
	12.0 m.n.													230	0·6	1·2	0·3	81
4	7.0 a.m.													350	8·4	0·8	0·6	84
	9.0 a.m.				44	3320	6160	61	8·4	27	620	70	10·9					
	11.0 a.m.													150	0·5	1·1	0·4	29
11	9.0 a.m.				44	3480	6250	66	8·2	18	620		10·7					

[1] The observations made on one control, at rest, and given the standard diet and fluid intake, are shown in this table. The absence of any abnormal changes in the constituents of the blood is to be noted. The large excretion of urine and the high concentration of chloride in the urine are contrasts to the low excretion found in the case. In columns under "Urine" the quantities in any one line represent the amounts passed during the time between the last and the given specimen.

TABLE IIIB.[1]
Oral Intake of "Control B".

Day of Observation.	Time.	Carbohydrate. (Grammes.)	Protein. (Grammes.)	Fat. (Grammes.)	Calories from Fluid Food.	Total Calories.	Volume of Water *plus* Water Content of Food. (Cubic Centimetres.)	Calcium. (Gramme.)	Sodium Chloride. (Grammes).
1	11 p.m. to 5 p.m. ..	119	36	50	506	1070	1640	1·00	2·40
	5 p.m. to 11 p.m. ..	75	16	25	291	589	950	0·60	3·09
	11 p.m. to 5 a.m. ..	0	0	0	0	0	0	0·0	0·0
	5 a.m. to 11 a.m. ..	84	20	27	347	659	710	0·62	3·06
2	11 a.m. to 5 p.m. ..	87	26	37	225	785	840	0·80	1·22
	5 p.m. to 11 p.m. ..	52	8	12	66	348	620	0·32	1·07
	11 p.m. to 5 a.m. ..	0	0	0	0	0	0	0·0	0·0
	5 a.m. to 11 a.m. ..	84	19	25	347	637	950	0·61	3·05
3	11 a.m. to 5 p.m. ..	87	24	37	224	777	860	0·73	2·00
	5 p.m. to 11 p.m. ..	84	20	26	281	650	950	0·62	1·96
	11 p.m. to 5 a.m. ..	0	0	0	0	0	0	0·0	0·0
	5 a.m. to 11 a.m. ..	52	10	13	66	365	450	0·30	1·47

[1] This table shows the contents of the standard diet.

as a guide to the treatment of internal hæmorrhage, especially if the physician is aware of the fluctuations caused by the changes in blood volume.

Plasma Protein.

In the present series most patients when admitted to hospital showed some deficiency in plasma protein (Cases II, IV, V and VIII); the average reading on their admission was 6·1%, whereas the average on their discharge was 7·1%. In some (Cases III and VII) the depletion was serious, readings between 4% and 5% being found. The two controls average 7·7% at the beginning of the control period and 7·8% at the end, three days later. The fall in plasma protein level may be due to a deficiency of protein in the diet and the prolonged loss which occurs during slow bleeding from an ulcer, for a massive hæmatemesis may be preceded by a slow loss of blood lasting for weeks or even for months. Finally, with the massive hæmorrhage there is a further loss of protein. This depletion of plasma protein may lead to œdema of the tissues owing to the lowering of osmotic pressure; thus the healing processes in the ulcerated area are impeded and the risk of post-operative complications after surgical intervention is increased. Two patients (Cases I and VII) were operated upon for the arrest of hæmorrhage, and both died several weeks later from intraperitoneal abscess caused by a leak at the site of the anastomosis. This may have been attributable to the poor healing power of the tissues.

In most cases in the present series the plasma protein maintained a level not much below normal, with slight fluctuations. In Cases III and VII, when the plasma protein level was very low and the intake by mouth was negligible, transfusion caused a slight rise; but a significant elevation of the plasma protein level appeared only when a liberal amount of protein was taken by mouth.

FIG. 4

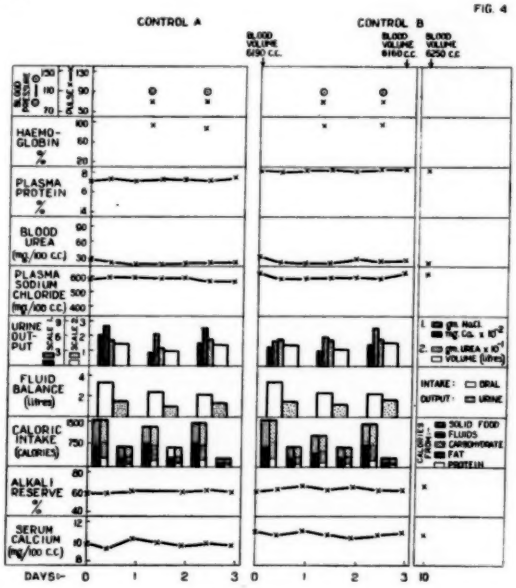

FIGURE IV.

Showing the observations made on two control subjects, at rest, and given the standard diet and intake of fluid. The absence of any biochemical changes is to be noted.

Holman and his co-workers,[7] in studies on dogs, have shown that there is a dynamic equilibrium between the tissue proteins and the plasma protein content, which depends on the physiological needs of the moment; that without food protein both the plasma and the tissue proteins are progressively depleted; and that the protein of the plasma may be relatively more depleted than that of the tissues. Finally, they showed that with adequate or high intake of protein by mouth both plasma and tissue protein levels are restored, this restoration being most conspicuous in the plasma.

After a hæmorrhage has occurred the protein lost could be restored by mobilization of the existing protein stores. We found, however, that after a severe hæmorrhage patients often have an intense distaste for solid or semi-solid foods and sometimes will take only water (Cases III, IV and VII). They are benefited by blood transfusion, which supplies protein as well as red cells and chlorides.

Blood Urea.

Several workers have shown that after gastro-intestinal hæmorrhage there is usually a rise in the level of the blood urea. Black[8] considers that this rise is due to a decreased renal blood flow. In the present series the blood urea level was found to be elevated in nearly all cases, varying from 43 to 240 milligrammes per 100 cubic centimetres of blood.

The blood urea level fell after the hæmorrhage had ceased, and this fall was hastened by the liberal administration of fluids by the mouth or rectum, or by the intravenous infusion of blood or glucose saline solution (Cases IV, VI, VII and VIII). When the fluid intake was restricted the excretion of urine was diminished and the blood urea level remained high (Case III). Most patients, when given liberal amounts of fluid, excreted larger amounts of urea than the two controls (Cases I, VII and VIII). However, this increased excretion of urea was due to the increased volume of urine excreted, the urea concentration usually being low in relation to the level of urea in the blood. In only one case (Case VII) was the urea concentration greater than 2% when the blood urea level was higher than 60 milligrammes per 100 cubic centimetres. In another case (Case III) it was as low as 0·5% when the blood urea level was 240 milligrammes per 100 cubic centimetres. Patients in the present series generally passed 20 to 30 grammes of urea per day when the blood urea level was falling, whereas the controls passed on an average 12 to 15 grammes of urea per day. Variation in the formation of urea is indicated by the fact that the rise and fall in the level of the blood urea did not always bear a direct relationship to the amount of urea excreted.

Drevermann[9] considers that the rise in blood urea level can be accounted for by the formation of urea from digested blood proteins, and by the diminished output of urine, which is a compensatory mechanism resulting from depleted tissue fluids. He was able to produce a rise in the blood urea level by introducing human blood into the stomach of a normal person.

McCance and Widdowson[10] have shown that salt deficiency is accompanied by a rise in the blood urea level, and they attribute this to diminished glomerular filtration and additional absorption of urea. The salt deficiency which was found in the present series may have contributed to the elevation of the blood urea level. It is difficult to assess the damage to the patient caused by the rise in the blood urea *per se*. It is of interest to note, however, that a fall in the blood urea level was accompanied by a general improvement in the condition of the patient.

FIG. 5

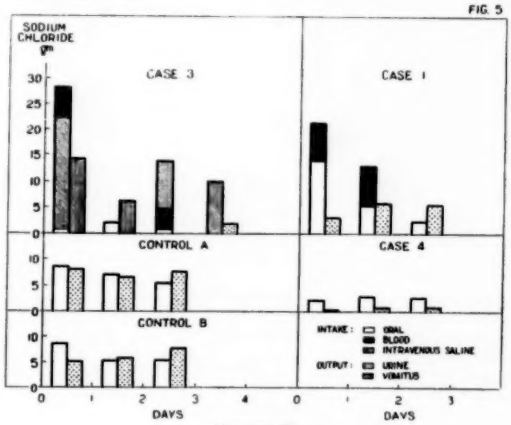

FIGURE V.

Showing the pronounced retention of sodium chloride observed in patients during the liberal administration of chloride by mouth or intravenously. This provides a striking contrast to the balanced intake and output of the controls. In Case III there was no excretion of sodium chloride in the urine till the fourth day. In Case IV sodium chloride in capsules was given by mouth, and in Case I additional sodium chloride was added to the food and drinks.

Sodium Chloride.

In practically all instances, when the patient was first admitted to hospital there was a marked decrease in the chloride content of the blood accompanied by the disappearance of chloride from the urine. In four of the more severe cases of hæmatemesis (Cases II, III, V and VII) the average plasma chloride content on admission to hospital was 470 milligrammes of sodium chloride per 100 cubic centimetres of plasma. "Control A" had a plasma sodium chloride content of 570 milligrammes per 100 cubic centimetres at the beginning of the control period and of 560 milligrammes per 100 cubic centimetres at the end. "Control B" had a plasma chloride content of 620 milligrammes *per centum*, which remained unchanged.

There are probably several factors which contribute to the fall in the plasma chloride content. First, during the period of partial starvation which often precedes and follows the initial hæmorrhage, there is a diminished intake of sodium chloride in the food; secondly, there is a further loss of sodium chloride by loss of blood into the alimentary canal; finally, vomiting, which commonly accompanies hæmorrhage into the upper portion of the intestinal tract, involves loss of gastric contents rich in sodium chloride.

The depletion of plasma chloride was associated with a greatly diminished output of chloride in the urine, and in those cases in which the plasma sodium chloride content was under 500 milligrammes per 100 cubic centimetres of plasma, it was common to find a complete absence of chloride in the urine (Cases II and III).

Falconer and Lyall[11] consider that the normal intake of sodium chloride in the diet is six to twelve grammes per day. In our series controls A and B consumed eight or nine grammes of sodium chloride per day, and excreted over 90% of this in the urine during the control period—a urinary chloride concentration ranging from 0·23% to 0·75%. This balanced intake and output of chloride in the controls provides a striking contrast to the retention of chloride observed in the patients during the liberal administration of chloride (Figure V). This retained chloride restores not only the plasma chloride content, but also the depleted content of the tissues. McCance and Widdowson[10] have shown that during artificially produced salt deficiency an enormous amount of sodium chloride is lost from the extracellular fluids, which is replaced when the deficiency is made up, and the symptoms experienced by these subjects (McCance[12]) closely resemble the symptoms of many of our patients.

We attempted to increase the chloride content of the blood in our patients by the addition of sodium chloride to the food and drinks. We also gave additional sodium chloride in capsules, each containing 0·5 gramme, which were more effective though the rise in plasma chloride level was slow (see Case IV). Transfusion also caused a moderate increase, though this was not so great as that obtained by the intravenous injection of saline solution (Cases III and VII).

At this stage one cannot assess the importance of the part played by salt deficiency in determining the outcome of severe hæmatemesis. It would appear to be rational to restore lost chloride by providing a liberal but judicious amount in the food and drinks; but in no circumstances should either be rendered unpalatable. The administration of gelatine capsules containing 0·5 gramme of sodium chloride may be a more effective method. Rectal injections of saline solution may be well tolerated, and finally the physician should infuse normal or hypertonic saline solution should the need be urgent. The addition of glucose (5% to 10%) to the infusion fluid aids nutrition.

Output of Urine.

Thirst, which is often intense, is a constant complaint from patients suffering from acute hæmorrhage from the stomach or duodenum. It is usually associated with a dry tongue and diminished output of urine (Cases III, IV and VII). With adequate replacement of fluid, thirst is slowly relieved, the tongue becomes moist and the output of urine increases. It is important not only to replace fluid in the dehydrated tissues, but also to hasten the elimination of waste products by producing a liberal diuresis, thus combating the rise in blood urea level and the tendency towards acidosis. In spite of the excellent teaching of Meulengracht, Witts and others, patients are still often kept for several days in a state of extreme dehydration.

Caloric Intake.

Meulengracht[13] has shown that the administration of food to a patient who has recently suffered a severe hæmorrhage from the stomach or duodenum does not tend to start a further hæmorrhage. Our studies suggest that a high caloric diet assists in restoring the plasma proteins (Cases V and VI) and chloride (Case I), as well as in contributing to the other needs of the undernourished patient. It may, however, be several days before the patient will agree to take semi-solid food. He is drowsy, nauseated, apprehensive and thirsty. His tongue is coated and dry. The initial craving is nearly always for frequent drinks of cold water. Later, sweetened fluids with a trace of sodium chloride will be taken (barley water, orange drinks *et cetera*). Finally, after a few days the Meulengracht diet can be taken with comfort. We have endeavoured to give our patients a high caloric diet from the time of their admission to hospital; but Cases II, III, IV and VII show that in the early stages patients cannot always take this diet.

Lack of appetite may be accounted for by the general disturbance of bodily function from anæmia and circulatory failure, by abnormal biochemical changes such as chloride depletion, uræmia and acidosis, by the gastritis which frequently accompanies peptic ulceration, and lastly by the presence of large quantities of blood in the bowel. Devermann[9] found that human blood introduced into the alimentary canal of a normal person caused colic.

Alkali Reserve.

The drowsy state into which some of the patients lapsed was usually associated with a varying degree of acidosis, as indicated by a decrease in the alkali reserve. This was accompanied by thirst, dehydration, nausea and often vomiting, by a rise in the blood urea level, and by a fall in the blood chloride content. Usually the acidosis was only of a minor degree, the alkali reserve being 45% to 50% (Cases II, IV, V and VIII); in the normal controls it was 58% to 66%. Occasionally, however, the level fell below 40% (Cases III and VII). With the cessation of hæmorrhage and the liberal administration of fluids and food, the acidosis was usually relieved (Cases IV, V and VIII). At the same time a general improvement took place in the patient's condition and the biochemical levels returned to normal. In Case III, in which there was possibly some preexisting renal damage, acidosis persisted until Hartmann's sodium lactate solution was administered intravenously.

Calcium.

The serum calcium content remained within normal limits in practically all cases. In one (Case VII) the serum calcium content was low on the patient's admission to hospital and later fell to 7·7 milligrammes per 100 cubic centimetres. After transfusion a slight fall in the calcium level usually occurred (Cases I, II and VI). It would appear that the body is readily able to mobilize calcium from the liberal stores in the body, or to utilize calcium administered in the food.

Treatment.

The present studies uphold the contention that patients who have recently bled copiously from the stomach or duodenum as a result of peptic ulceration, are not only suffering from anæmia, but also from depleted blood volume, starvation, dehydration, chloride and protein deficiency, uræmia and acidosis. The objects of treatment should thus be: to arrest hæmorrhage and minimize the likelihood of further bleeding; to restore the blood volume and lost tissue fluids and relieve anæmia; and, finally, to supply adequate amounts of water, carbohydrates, proteins, chlorides and vitamins by mouth, and perhaps by the rectal or intravenous routes.

The patient should be placed at complete rest in bed. Morphine allays the patient's fears and gives him rest and sleep. With the liberal administration of fluid, however, less morphine will be needed. Alkaline powders and atropine still deserve a place in treatment.

Should hæmorrhage recur in spite of two or three blood transfusions, the surgeon may consider that operative interference gives the patient the only chance of survival. The dangers which beset surgical arrest of hæmorrhage are becoming less formidable now that the patient can be aided by massive blood transfusion and other restorative measures.

The method of feeding advocated by Meulengracht is rational; but, as has already been stated, it may be impossible to entice patients to take semi-solid food by mouth for hours or even days after the hæmorrhage has occurred. Fluids should be allowed in small quantities given frequently. At least two litres should be given per day in the form of citrated milk, barley water, orange drinks and broth. Should the toxæmia and dehydration be severe, or should nausea and vomiting prevent adequate administration of fluid by mouth, resort should be had to the intravenous or rectal route. The rectal administration of normal saline solution may be well tolerated for one or two days, and may be useful in combating both dehydration and chloride deficiency. The method has its limitations, however, because many patients soon become intolerant to rectal injections. Moreover, protein and carbohydrate are not absorbed when given *per rectum*.

It is best to give these toxæmic, dehydrated and anæmic patients a blood transfusion followed by the slow intravenous infusion of 5% glucose in normal saline solution (100 to 150 cubic centimetres per hour); when the chloride content is greatly depleted one or two litres of 3% saline solution may be given. Blood transfusion may also be given if the patient is suffering from prolonged circulatory collapse, as indicated by fainting, sweating, poor volume of the pulse and a systolic blood pressure below 90 millimetres of mercury; secondly, if the hæmoglobin level falls below 40% as a result of acute hæmorrhage; or lastly, as a preliminary to surgical operation for the arrest of hæmorrhage.

Should a transfusion be decided upon, it is usually best to give two or three pints (1,200 to 1,800 cubic centimetres) of blood. It should be run in at the rate of 150 to 300 cubic centimetres per hour unless the patient is suffering from acute blood loss, in which case a more rapid rate may be necessary to preserve life. If an operation is to be performed for the arrest of hæmorrhage, the hæmoglobin value should be elevated to at least 60% before operation. This is nearly always possible, because the bleeding from a peptic ulcer tends to be intermittent. Slow drip blood transfusion should be continued during the operation and for some time after the patient returns to the ward. This enables a skilled surgeon to perform a radical operation, such as partial gastrectomy, with very little risk of death from surgical shock. The greatest danger lies in the delayed complications, such as perforation, perigastric abscess, hæmorrhage and ileus (Cases I and VII).

Summary.

1. A detailed investigation was undertaken of the biochemical changes which occur as the result of massive hæmorrhage from the stomach and duodenum. Eight cases were studied in detail, and occasional determinations were made in five others. Two normal controls were investigated over a period of three days.

2. When the patients were not only exsanguinated, but were drowsy, nauseated, thirsty and dehydrated, it was found that the pulse rate was rapid, the blood pressure and hæmoglobin value were low and the blood urea level was elevated, and there was a fall in the blood chloride and plasma protein content and in the alkali reserve. The urine was diminished in amount and the chloride content was greatly lowered.

3. The effects of the administration of food and fluid by mouth, the rectal injection of normal saline solution and the intravenous infusion of citrated blood and glucose saline solution are discussed.

4. The blood urea level fell after the hæmorrhage had ceased, and this fall was hastened by the liberal administration of fluid. When the fluid intake was restricted the excretion of urine was diminished and the blood urea level remained high. Most patients, when given a liberal amount of fluid, excreted larger amounts of urea than the two controls. However, this increased excretion of urea was due to the increased volume of urine excreted, the urea concentration usually being low in relation to the level of urea in the blood.

5. The place of surgery in the arrest of hæmorrhage is considered, and the risk of delayed post-operative complications is emphasized.

Acknowledgements.

We are deeply indebted to Mr. H. F. Holden for his generous assistance and advice throughout our investigations. Our sincere thanks are also due to Dr. C. H. Kellaway, Director of the Walter and Eliza Hall Institute, and to members of the medical staff and dietetic department of the Royal Melbourne Hospital.

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Reports of Cases.

MULTIPLE LUNG ABSCESSES.

By HOWARD J. EDELMAN, M.B., B.S. (Adelaide),
House Surgeon, Royal Adelaide Hospital.

IN most instances abscesses of the lung occur singly; the case reported below derived its main interest from the simultaneous occurrence of a few large and many small abscesses in both lungs. The fact of good recovery is also noteworthy.

In the consideration of pulmonary abscesses the following classification seems to be quite satisfactory: (i) infections from within the bronchial tree, notably the various types of pneumonia, and aspiration of foreign bodies and of food matter during operations on the mouth or upper respiratory tract; (ii) infection from without the bronchial tree, which may be (*a*) hæmatogenous, as illustrated by septic emboli, (*b*) lymphatic, as the spread of carcinoma, and (*c*) occurrence by direct extension from adjacent suppuration, such as empyema and subphrenic abscess.

Clinical Record.

The patient, an unmarried female, aged fifty-four years, was admitted to hospital on January 22, 1940, with a history of difficulty in breathing and pain in the right side of her chest of three days' duration. She had had several rigors at the onset of the attack and had been vomiting frequently since then. She had no cough. In October, 1939, the patient had had a radical mastectomy for carcinoma of the right breast. This was followed six weeks later by a course of deep X-ray therapy.

The patient was a rather thin woman; her face was very flushed, but not cyanosed, and her expression was anxious and weary. Her temperature was 100·8° F., her pulse rate was 116 per minute, the respirations numbered 24 per minute, and the systolic blood pressure was 110 and the diastolic pressure 65 millimetres of mercury.

The chest signs were as follows: impairment of percussion note and decrease in the intensity of breath sounds over the bases of both lungs posteriorly and also in the lower part of the right axilla. Crepitations could be heard at the bases of both lungs posteriorly.

A diagnosis of bronchopneumonia was made, and routine treatment was instituted. In addition, "M & B 693" was given for seven days. For the first week the patient was miserable and depressed and vomited frequently. The temperature ran an uneven course between 99° and 101° F., and the respirations were rapid; the physical signs remained practically unchanged. Towards the end of the second week the temperature began to settle and the patient's general appearance improved, although she was still very depressed.

On February 12, 1940, her temperature having risen over the previous two days, she complained of pain in the left side of the abdomen. She was tender in the left flank and pus was present in the urine. The lung signs were slight; there were only a few râles at the base of the left lung. Culture from the urine yielded a growth of *Staphylococcus aureus*. A blood examination on January 27, 1940, gave the following information: the erythrocytes numbered 3,800,000 and the leucocytes 9,250 per cubic millimetre; the hæmoglobin value was 13·26 per 100 cubic centimetres (85% Sahli).

On February 15, 1940, she appeared much better, but still complained of pain in the chest. The temperature was still between 99° and 101° F. An X-ray film of the chest was taken, and this revealed several abscess cavities in both lungs. On receipt of the X-ray film the chest was examined very carefully, and a small patch of amphoric breathing less than two inches in diameter was detected at the angle of the right scapula. This was the only sign of cavitation discovered.

Postural drainage was begun, and the patient began coughing up sputum in moderate amounts. For the next fortnight she expectorated copious quantities of foul yellowish brown sputum. An examination of the sputum revealed both Gram-positive and Gram-negative cocci, and no tubercle bacilli; leucocytes and erythrocytes were present, but no elastic tissue.

On February 23, 1940, a friction rub was heard in the lower part of the right axilla. During the next two days an abscess developed on the right heel. This was incised and the pus on culture yielded a growth of *Staphylococcus aureus*. At this stage bronchoscopic drainage was considered, but was deemed inadvisable because of the good response to postural drainage and the thinness of the abscess walls. The signs of cavitation became rather more definite, and further X-ray films revealed increase in size and coalescence of the cavities.

On March 8, 1940, the patient began to feel much better, and her appetite, which had been poor throughout, improved. An X-ray film taken on March 18, 1940, showed that there had been a rapid resolution of fluid and a decrease in size of the cavities. The clinical improvement continued and the sputum became scanty. On March 29, 1940, examination of the chest revealed no signs of cavitation, but the percussion note was impaired, the breath sounds were decreased, and râles were present at the base of the right lung. Further X-ray films revealed a continuation of the process of resolution; the last film, taken on June 11, 1940, revealed complete resolution except for a small patch of infiltration at the base of the right lung.

On May 4, 1940, the patient was transferred to a convalescent ward situated in the foothills, where she stayed and maintained her improvement until June 7, 1940; she then had a "flare-up" of acute pyelitis. The right kidney was palpable, and the urine still infected with *Staphylococcus aureus*. After ineffective treatment with milder drugs a course of "Uleron" tablets was begun on July 4, 1940. The urine was sterile on July 15, 1940, and the "Uleron" treatment was discontinued on July 17, 1940. Since then her condition has been improving constantly, and she was transferred to a convalescent hospital at the seaside on August 16, 1940.

Summary.

A case of multiple lung abscesses has been described. The ætiology is doubtful. The following may be suggested: (*a*) congenital cystic lungs—this is possible, although it cannot be proved clinically; (*b*) embolic abscesses—this theory is supported by the occurrence of other infections, such as pyelitis and a subcutaneous abscess on one heel.

A feature worthy of note is the satisfactory emptying and resolution of all abscess cavities, the only treatment being postural drainage and the administration of an expectorant mixture containing creosote.

In addition to its unusual interest, this case also serves to illustrate the fact that pulmonary abscess is not a rare complication of the various types of pneumonia.

Acknowledgement.

I should like to thank Colonel A. R. Southwood, honorary physician, Royal Adelaide Hospital, for many helpful suggestions.

Reviews.

ANATOMY OF ANIMAL TYPES.

THE constant stream of medical and other zoology students through our universities has resulted in a second edition of Briggs's hand-book devoted to the anatomy of the animal types likely to be used in first-year classes.[1] The first edition was published in 1934.

The work has been increased in size by about fifty pages, which is largely due to a new chapter on the dissection of the sheep's heart, and the rewriting of certain chapters.

The author still feels that illustrations should be excluded in view of the fact that the book is intended as a guide to the dissector and that he should be forced to "verify for himself statements contained in the text".

This viewpoint might be debated; but on the other hand the descriptions given by the author are particularly clear and, so far as we can judge, the student should have no difficulty in carrying out his work. As an aid to laboratory work in university classes where demonstrators may be called on when necessary, the work is excellent.

AN ATLAS OF HISTOLOGY.

THE book by Burton Bradley entitled "Elementary Atlas of Histology", is another indication of the advancing tide at long last of Australian publication.[2] It is unusual in style, for the work consists of 58 plates and 58 descriptions of those plates. The plan is thus almost the opposite of that taken by Dr. Briggs in the volume referred to above. The aim is to help the beginner in histology to understand what he sees under the microscope.

Dr. Bradley suggests that the photographs and also the very complete (and usually composite) drawings found in most text-books of histology are difficult for the beginner. The photograph is probably less clear than the preparation which the student is studying, whilst the drawing contains the details collected from hundreds of observations, and is too complete, too perfect.

The illustrations in this book are half-tone reproductions of freehand drawings of the preparations usually seen in ordinary practice. The author is very careful to point out that the illustrations are certainly not to act as a substitute for the examination of actual specimens.

The idea is novel so far as an atlas of histology is concerned, and has much to recommend it, though it might be more emphasized that use in conjunction with a modern text-book of histology is desirable. For example, it is very difficult to see how a beginner could understand the section through a hairy skin (plate 16) unless he saw a diagrammatic section which did pass through the long axis of a hair and follicle. It is quite true that ordinary sections are usually much more haphazard than the diagrammatic figures illustrating whole structures; but one sort of figure is complementary to the other.

The drawings (which are clear and well done) will in fact be very interesting to use in conjunction with some of the fuller illustrations of well-known books.

One improvement which might well be made would be a better guide to the different portions of a section. To find the reference letters in plate 45, for example, is almost a puzzle. There are several small points which need correction. Thus the spireme stage in mitosis is now generally regarded as suspect. The book may be regarded, however, as a very useful atlas for beginners in histology, and as an adjunct to the more usual text-books which will be necessary.

The publishers are to be congratulated on the excellent reproduction of the illustrations.

[1] "Anatomy of Animal Types for Students of Zoology", by E. A. Briggs, D.Sc.; Second Edition, revised and enlarged; 1940. Sydney: Angus and Robertson Limited. Demy 8vo, pp. 310, with illustrations.

[2] "Elementary Atlas of Histology", by Burton Bradley, M.B., Ch.M., M.R.C.S., L.R.C.P., D.P.H., with a foreword by J. B. Cleland, M.D.; 1940. Australia: Angus and Robertson Limited. Crown 4to, pp. 122. Price: 15s. net.

The Medical Journal of Australia

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PHYSICAL FITNESS AND ITS DETERMINATION.

THESE are times when the term "physical fitness" is on almost everybody's lips, times when ability to live strenuous days, to do a man's work and more, is regarded as almost the *summum bonum* of life. Even before the war perfection of form, muscular development and powers of endurance were set up as goals that made any effort worth while. Young men and women were prepared to deny themselves luxuries or pleasures that might make them "soft", and those of older generations sometimes tried to follow their example. In some countries beauty of the body seemed to be almost an end in itself, so persistent and intense was its pursuit. Naturally enough, youth was proud of all that it had achieved and lost no opportunity of displaying either its comeliness or its proficiency. Now that the nations are at war and the British peoples are fighting for their very existence, culture of the body takes on a new significance. All that the human body can do is being put to the service of the State, and both men and women are using in the cause of humanity all that they have gained for their bodies by steady application and training. In the present circumstances, therefore, we need not concern ourselves with physical fitness in regard to conditions as they were before the outbreak of war.

It will be quite obvious that our conception of the need for physical fitness during war-time will vary according to the duties that we are to be called upon to perform. In other words, when an examinee is facing a medical examiner, the examiner must know for what type of action his fitness is required. Sailors on the decks of a warship or in a submarine, infantrymen in the front line, and air force pilots need, generally speaking, to be fitter than men from the three services occupied solely on shore, at the base or on the ground. At the same time, different degrees of fitness will meet the needs of those employed in industry, whether it be in the manufacture of munitions or in other essential services. Recruits for the army, like recruits for the other branches of the defence forces, are submitted to certain examinations by medical practitioners in order that only those who attain a certain standard shall be accepted. The history of the Australian Army Medical Corps in the war of 1914-1918, as set out by Colonel A. Graham Butler in his well-known book, has shown in the most convincing way possible that the maintenance of a high standard of fitness among recruits accepted for service and the safeguarding of the health of those in the field contributed more than anything else to the high and enviable standard of efficiency and performance of the Australian Imperial Force. On many occasions the need for care in the examination and acceptance of recruits has been emphasized, and the matter should not call for further discussion; but the receipt from the Director-General of Medical Services of a report from medical officers of the Hospital Ship *Manunda*, published in this issue, makes further reference desirable. We do not propose to take readers over the details of the report; they should study the document with care, taking each section separately and forming their own conclusions about it. Though, as the authors of the report point out, the number of men returned to Australia on medical grounds as unfit for service is comparatively small, we are not altogether inclined to agree that this is "a tribute to the judicious care in the early medical examination". Admittedly more men would have been sent home if examiners had been more lax than they were; but men were sent home who should never have gone away from Australia. The statements in the report should rather be used as a stimulus to a deeper probing for tell-tale facts and to greater care in future examinations. Men anxious to enlist will sometimes withhold information about past illnesses or other facts that might reveal possible causes of subsequent breakdown; the truth can often be discovered, and great diligence should be shown in attempts to discover it. Sometimes grave cause for doubt regarding acceptance may be present, and sometimes there may be merely grounds for suspicion. To accept a recruit merely to "do him a good turn" is most reprehensible; it puts an unnecessary burden on the State financially and otherwise, and may be a cause of danger to other members of the forces who are not unfit.

In turning to the question of physical fitness in industry, we would draw attention to a paper that has recently been published by H. H. Kessler, of Newark, New Jersey.[1] Kessler discusses the determination of physical fitness from both the military and the industrial points of view, and it will be of interest to Australian medical practitioners to know that his is only one among many recent references in American medical journals to the programme of "medical preparedness" for a national emergency. For the sake of simplicity, and having already discussed the military side, we must be content to refer only to some of Kesslers' remarks that are applicable to industry. His point of view is refreshing. He regards physical fitness as a socio-economic concept involving the social evaluation of a series of anthropological, physiological and psychological traits, and he points out that it becomes a medical concept in the presence of a physical defect. False concepts of capacity to work have, he thinks, created vague standards of physical fitness that have condemned the physically defective who are handicapped by minor disabilities, but who possess "great potentialities for functional performance". One of their effects has been the setting up

[1] *The Journal of the American Medical Association*, November 9, 1940.

for the physically useful person of "an arbitrary industrial age deadline of 40"; they have also given excessive weight to psychological and aptitude tests for the determination of physical fitness, while human energy and capacity have in general been largely underestimated. By the science of pathology it is possible to determine the nature of an illness or defect, but not the extent of the remaining health or the extent of adaptation of the remaining functions. "A person lives not only with his pathologic lesion but also with the remaining undisturbed organs and functions." Defining physical fitness as the ability to perform productive and continuous work, and stating that it is based on native ability, training and the presence or absence of illness, Kessler insists that the only value of physical examination in the determination of these factors is its possible detection of gross illness or profound emotional instability. Static defects should, he thinks, be separated from illness states and their limited effect on working capacity properly evaluated. By static defects he means such defects as short stature, underweight, a stiff elbow, loss of a finger or leg by amputation, leg shortening, a paralysed hand, and so on. This means that we have to put the right man in the right place, or, what comes to the same thing, choose the job that will suit the man. Many branches of industry are unsuited for men with static defects, but in certain industries and even in some essential services jobs that can be done by such persons can surely be found. At the present time there should be work for everyone, and it frequently falls to medical practitioners to determine the fitness of a man for certain work. If, as Kessler states, the average man can do the average job, the doctor who undertakes the physical examination of employees will find it easier to make a decision on that account. And in making up his mind about a pathological condition he must try to estimate the remaining health and the extent of adaptation of the remaining functions—he has to estimate both disease and health.

What has been written about industry brings us once more to the determination of physical fitness for the armed forces. Here the job is strenuous and exacting, and its nature will not change; there is only one standard for front line service, and that is one that has as its basis freedom from all bodily and mental disease—the job cannot be chosen to fit the man; the man must fit the job.

Current Comment.

TETANUS IMMUNIZATION.

THE death rate from tetanus among the men on active service in the war of 1914-1918 gave lasting impetus to the effort to find some means to prevent this disease. The immunity that can be conferred on a person against an infective disease is of two types, passive and active. Passive immunity is conferred by injecting into the person antibodies against that disease, and without effort on the part of the person some degree of immunity is established, but only until the injected antibodies are destroyed or excreted. Active immunity, on the other hand, is immunity developed by the body itself; if toxins are injected into a person, or if the person contracts the disease, then his own tissues produce antibodies to protect him from the toxins that have been introduced. This is Nature's method of immunization, and is permanent, not because the antibodies produced last forever, but because with most diseases, once a person's tissues have produced these substances, they are able to do so again much more rapidly and effectively when at any time in the future the necessity arises. Tetanus antitoxin, given soon after a wound occurs, confers a temporary passive immunity, and has saved many lives. It may be noted here that in the presence of a virulent infection the dose of 500 to 1,000 units so often given is frequently insufficient to prevent the onset of the disease. Ramon, of the Pasteur Institute, in 1926 reported satisfactory active immunity against tetanus by the use of formol toxoid or anatoxin, a direct sequel to his work on diphtheria immunization. It will be remembered that active immunity to diphtheria was first produced by von Behring by the use of a mixture of toxin and antitoxin (see THE MEDICAL JOURNAL OF AUSTRALIA, October 12, 1940). The toxin caused the body to react immunologically as it would to the disease itself and to develop an active immunity, the antitoxin meanwhile providing a temporary passive protection from the morbid effects of the toxin. Ramon found that the toxins of diphtheria and tetanus, when treated with formaldehyde and heat, became non-toxic, but retained their power to stimulate an active immunity. These detoxified products were called anatoxin by the French, but are now referred to as toxoid in English-speaking countries. If precipitated with alum they become relatively insoluble, with the advantage that they are slowly absorbed and unpleasant reactions are thus diminished.

Commander W. W. Hall, of the United States Navy Medical Corps, has recently reported the results of tetanus immunization of the men of that navy.[1] A preliminary investigation revealed that alum-precipitated toxoid was a much more effective immunizing agent than plain toxoid in that it produced uniformly higher titres of antitoxin in the patients' blood. It was also found that the longer the interval, up to eight weeks, between the injections, the better were the results obtained, but that immunization with intervals of only two weeks gave considerable protection. In one trial group, the first injection caused a very slight rise in serum antitoxin. The second injection eight weeks later caused a considerably greater rise, followed by a slight fall in the ensuing months. A third injection nine months later produced a rise much greater than any previous one. All the men included in this survey, and they numbered 3,446, produced a satisfactory titre of serum antitoxin. It must be remembered, however, that the antibodies circulating in the blood stream are not the most significant part of the protective mechanism of active immunity, nor perhaps are they a satisfactory measure of the degree of that immunity. The really significant thing is that once a person is actively immunized, be it by an artificial process or by the disease itself, the antibody-producing power of his tissues is tremendously increased, and he becomes able quickly and effectively to combat the infection if he meets it. The most striking tribute that can be given to tetanus immunization is, Hall tells us, and we presume he is correct, that from among the thousands who have been immunized in the United States Navy and in the armies of Britain, France and Italy no case of tetanus has yet been reported. The present plan of immunization in this navy consists of two injections eight weeks apart as basic immunity, an injection at the time of injury if deemed necessary to raise the titre of blood antitoxin rapidly, and an injection each four years after the basic immunization to maintain immunity at a high level. Alum-precipitated toxoid is used for all injections and the dose for each is one cubic centimetre.

The reactions produced by this routine are very slight. Soreness of the arm is almost invariably gone in two days. General reactions seldom make it necessary for men to report sick. It is found that intramuscular injection causes less local reaction than if the hypodermic site is used, but serum antitoxin levels are just as high. Care in the preparation of the toxoid minimizes the reaction produced. A certain brand of toxoid was found to be producing reactions, in one case frank anaphylactic shock, after the

[1] *Annals of Internal Medicine*, October, 1940.

second injection. Analysis and investigation showed that it contained a protein substance because it had not been washed with sufficient thoroughness during preparation, and this caused sensitization and subsequent reactions. The sensitization produced by plain toxoid and alum-precipitated toxoid was then studied in guinea-pigs, animals which show very pronounced hypersensitivity phenomena, and it was found that the process of alum-precipitation, if carefully done and accompanied by thorough washing, yields a product much safer than plain toxoid.

Tetanus immunization with toxoid is at present being undertaken among the armed forces of the Commonwealth of Australia; from this we shall be able, when we have respite, to form further estimates of its value. In any case its adoption in disciplined groups other than the armed forces seems inevitable. Unfortunately the barbarism of modern warfare has rendered the civilian as liable to injury as the soldier, and the civil population is not a controlled group. The responsibility for the protection of the members of the community from tetanus, as from any other preventible disease, rests partly with themselves and partly with their medical officers, both private and state. Even in Australia this responsibility may become an urgent one, and medical practitioners will do well to pay special attention to this subject.

THE VALUE OF WHOLEMEAL.

EXPERIMENTS showing more convincingly than ever the superiority of wholemeal over white flour have been carried out by Harriette Chick at the Lister Institute in the Division of Nutrition, formerly of London, but now temporarily housed at Cambridge.[1] The experiments were carried out as a result of a proposal made by the Ministry of Food to reinforce the white flour used in the making of bread by the addition of vitamin B_1 in order to make its nutritive value more nearly equal to that of wholemeal. Chick reminds her readers that white flour contains much less vitamin B_1 than wholemeal flour, but that this is only one of a series of relative defects. The Accessory Food Factors Committee of the Lister Institute and the Medical Research Council have enumerated these defects and have recommended that flour used for the making of bread should contain at least 80% to 85% of the wheat grain, the outer layers of the bran and cuticle only being rejected. White flour is not only poorer than wholemeal flour in vitamin B_1, but it also has a lower content of the vitamins in the B_2 series, minerals and protein. In these circumstances it seemed to Chick that it was worth while to compare by experimental methods the nutritive values of white and wholemeal flour when the deficiencies of the former in vitamin B_1, in fat-soluble vitamins, in protein and in minerals were made good, but when any difference in vitamin B_2 was not corrected.

Rats were used for the experiments. Two diets were arranged, consisting as largely as possible of white flour and wholemeal flour respectively, with the addition of a salt mixture to remedy the known defects of cereals in this respect and of extra protein in the form of casein to bring the amount of protein in the diet up to the optimum proportion for the growing rat. Pure vitamin B_1 was added to the white flour diet in optimum amounts. Chick sets out the percentage composition of the two diets, and shows her results in a table and by means of a graph. During the first two weeks of the experiment the average weekly increase in weight of animals on the white flour diet (11·8 grammes) was about half of that of animals on the wholemeal diet (22·8 grammes). The food intake of animals in the former group was also less, the average being 35·3 grammes of food (dry weight) as compared with 53·2 grammes of those on the wholemeal diet. The digestibility of the white flour diet was superior, but the utilization of the assimilated food was inferior. When allowance was made for loss in the fæces, it was found that 2·91 grammes of the white flour diet were required to produce an increase in weight of one gramme, but only 2·13 grammes of the wholemeal diet. At the end of the second week of the experiment the diets of the two groups were changed. The animals previously receiving a white flour diet now received a wholemeal diet and made an immediate spurt in growth, the average weight increasing each week by 24 grammes; those that had received wholemeal now received white flour and their growth was immediately checked, the average increase in weight falling to about seven grammes a week.

Chick attributes the inferiority of white flour as compared with that made from wholemeal to a shortage of the B_2 vitamins. She adds that further experiments are now in progress to determine in which constituent of the vitamin B_2 complex white flour is most seriously deficient. Few medical investigators will doubt that experimental work of this kind is applicable to man and the results of Chick's future work will be awaited with interest. The time may yet come when the manufacture of bread from white flour as we know it will be prohibited.

[1] *The Lancet*, October 26, 1940.

AMERICAN MEDICINE AND THE WAR.

IN view of the recent utterances of the Prime Minister of Great Britain, British people in every part of the Empire are gravely concerned with the attitude of the United States of America to Great Britain and the war. In any national upheaval the part played by the medical profession is of the greatest importance, and it is possible to see in the actions of organized medicine some indication of the attitude of the general community. When, therefore, we read in *The Journal of the American Medical Association* of November 9, 1940, an editorial article headed "Medicine Organizes for the Emergency" we may take heart in the knowledge that America realizes her own danger as well as ours. This editorial article, which, it should be noted, refers to "the emergency" and not "an emergency", draws attention to a statement in another part of the issue emanating from the Committee on Information of the Division of Medical Services of the National Research Council and describing the organization of various committees and subcommittees appointed to deal with medical problems associated with the military and naval medical services. In the special statement on medical preparedness it is pointed out that the National Academy of Sciences was established by President Lincoln in 1863 that it might, when called upon, give advice to the government on "any subject of science or art". During the Civil War the academy dealt with both military and naval problems. In 1916 the academy offered its services for national defence, and at the request of President Wilson created the National Research Council as its active agent in the organization of the scientific resources of the country. During the war years it became clear that the council might be suitable for the carrying out of certain work in times of peace. In 1918, therefore, President Wilson issued an order requiring the National Academy of Sciences to perpetuate the National Research Council and outlining its duties. These included not only the investigation of problems connected with defence, but also research in the several branches of science. Since the war of 1914-1918 its activities have extended chiefly into the latter sphere.

The committees and subcommittees already mentioned have been formed as the result of a request from the United States Army and Navy Medical Corps for assistance in the standardization of certain medical phases of its work. The committees are devoted to every branch of medical science, and in addition there is a committee on information. This committee has appointed two subcommittees, one on publicity and the other on hospital records. While Australian medical practitioners welcome the formation of these committees as a sign of American vitality, they also realize that the committees will collect information that will be of the greatest possible use to medicine wherever it is practised.

Abstracts from Medical Literature.

THERAPEUTICS.

Sulphathiazole.

W. H. SPINK AND A. E. HANSEN (*The Journal of the American Medical Association*, September 7, 1940) describe the uses of sulphathiazole. This drug is rapidly absorbed and rapidly excreted in the urine. In pneumonia the doses used are two to four grammes followed by one gramme every four hours until the temperature remains normal for forty-eight hours. Many cannot take the drug by mouth, in which case a 5% solution in distilled water is given intravenously, three grammes as an initial dose and then one gramme every eight hours until oral therapy can be commenced. The clinical effect is less than that achieved with sulphapyridine, vomiting is rarer, but dermatitis in papular or nodular form is more frequent. Staphylococcal infections have been treated with sulphathiazole; wound infections, *otitis media*, carbuncles, ulcers, osteomyelitis and subcutaneous abscesses responded fairly well, especially when sulphathiazole was applied to the wound. The best results were obtained in staphylococcal septicæmia; 15 patients recovered out of 15 treated. A concentration of 5% in the blood stream was aimed at and was thought to be effective. Patients suffering from urinary infections with streptococci, staphylococci, *Bacillus proteus* and *Escherichia coli* were treated, one gramme being given four to six times a day for seven to ten days. Improvement was obtained in 13 of 20 patients, many of whom had been treated unsuccessfully with sulphanilamide and sulphapyridine. The highest incidence of toxic effects was found in this group. The toxic effects generally were as follows: among 100 patients nine had dermatitis, six nausea and vomiting, one hæmaturia, two drug fever, two itching eyes, one anæmia, one nitrogen retention. The dermatitis was maculo-papular usually, but in four cases *erythema nodosum* occurred. On the whole, sulphathiazole was thought to be effective in pneumococcal infections, less nauseating than sulphapyridine, but more likely to cause a rash, more effective in staphylococcal septicæmia, and sometimes effective in varied urinary infections.

Peptic Ulcer.

I. CHASNOFF, S. LEIBOWITZ AND R. SCHWARTZ (*The American Journal of Digestive Diseases*, September, 1940) report the results of treatment with the Meulengracht régime in bleeding peptic ulcer. Meulengracht stated that he gave 2,300 Calories as follows: 6 a.m., tea, white bread and butter; 9 a.m., oatmeal, milk, white bread and butter; 1 p.m., unrestricted amounts of meat balls, *timbale*, broiled chops, omelette, fish balls, gratin of fish, vegetable or meat, mashed potato, puréed vegetables, cream of vegetables, vegetable soup, stewed apricots, apple sauce, gruel, rice and tapioca pudding: 3 p.m., cocoa; 6 p.m., white bread and butter, sliced meats, cheese and tea. In addition a teaspoonful of a powder containing sodium bicarbonate 2·0 grammes, magnesium subcarbonate 2·0 grammes, extract of hyoscyamus 0·1 gramme, ferrous lactate 0·5 gramme, was given three times daily after meals. The authors adopted this régime for 21 patients, five of whom required transfusions. Seventy-two controls were studied; their treatment consisted of starvation up to several days, with infusions, followed by minimal feedings in slowly increasing amounts. In both groups about four days elapsed between the onset of hæmorrhage and admission to hospital. One patient out of 21 treated by Meulengracht's method died, whereas eight of the control group died. On the other hand, perforation occurred in two of the Meulengracht group and only one of the control group. The patients on the Meulengracht régime felt well. The time in hospital was not lessened, being 26 days on the old Sippy diet and 34 days on Meulengracht's régime.

Cardiac Dropsy.

MAX WINTERNITZ, of Prague (*The Lancet*, May 11, 1940) describes the use of mercurial diuretics and urea in cardiac failure. By means of mercurial diuretics, such as "Novurit", "Esidrone", "Mersalyl" or "Neptal", injected intravenously or intramuscularly, life has been prolonged and made more comfortable for patients who suffer from congestive cardiac failure and dropsy. Eventually frequent injections of these drugs become irksome. Ammonium chloride, four to six grammes a day, on the day preceding and the day of injection sometimes enhances the effect of the injections; but the use of urea in doses of 20 grammes a day has been more effective in some cases and has produced adequate diuresis for long periods without the need for mercurial injections. The urea is dissolved in a single cup of strong unsweetened coffee, which covers the taste better than fruit juices. In two of the author's cases the urine output on this régime rose from 700 to over 1,000 cubic centimetres a day and continued so for two months.

Vitamin Therapy.

T. D. SPIES, D. P. HIGHTOWER AND L. H. HUBBARD (*The Journal of the American Medical Association*, July 27, 1940) describe recent advances in vitamin therapy. Vitamin B_1 (thiamine hydrochloride) relieves the pathological condition of beriberi, alcoholic addiction, pellagra and pregnancy. It increases the appetite and strength in subclinical beriberi. Nicotinic acid cures the glossitis, stomatitis and dermal lesions of pellagra, and relieves the early symptoms of that disease, loss of weight and strength, lassitude, abdominal pain, numbness, nervousness and forgetfulness. Riboflavin cures lesions at the corner of the mouth, shark-skin nose and conjunctival irritation and keratitis when due to deficiency of that substance. Vitamin *K*, essential for the maintenance of a normal concentration of prothrombin in the blood, is useful in jaundice to prevent excessive bleeding, and in hæmorrhagic disease of the new-born. One to five milligrammes given by mouth each day is effective if given for three or four days. In obstructive jaundice one or two ounces of bile, or the equivalent amount of animal bile salts, are necessary in addition, as the vitamin is ineffective if bile is absent from the intestine. Vitamin B_6 (pyridoxin) relieves nervousness, insomnia, weakness, muscular rigidity and awkwardness in walking in certain cases. Intravenous therapy with this vitamin has been followed by improvement in epilepsy, amyotrophic lateral sclerosis and *myasthenia gravis*. Spies reports improvement also in arteriosclerotic and post-encephalitic Parkinsonism. Jolliffe, on the other hand, reports no improvement in 80% of cases, but alludes to dramatic improvement in 20% of patients with non-post-encephalitic Parkinsonism who had been helpless for less than one year. Vitamin *E* (α-tocopherol) is said to benefit patients with amyotrophic lateral sclerosis. Vitamin deficiencies are usually multiple. Treatment includes rest in bed, a well-balanced diet of 4,500 Calories, rich in protein, minerals and vitamins. Dry powdered brewer's yeast, 75 to 100 grammes, liver extract, 75 to 100 grammes, or wheat germ, 150 grammes a day, should be given orally as supplementary to diet. Yeast and wheat germ contain nicotinic acid, vitamin B_1, riboflavin and vitamin B_6. In more severe cases of vitamin deficiency specific treatment with the isolated specific vitamins is necessary. The authors emphasize the importance of full investigation to exclude other causes of symptoms before the assumption is made that they are due to vitamin defect.

NEUROLOGY AND PSYCHIATRY.

Acute General Paresis.

M. HERMAN AND M. P. ROSENBLUM (*The American Journal of Psychiatry*, May, 1940), in studying the acute variety of general paresis, claim that it may manifest several clinical pictures, namely, (*a*) the fulminating type, (*b*) the convulsive type, (*c*) the catatonic type, and (*d*) the acute confusional type. Clinical histories are given to illustrate these types of onset, in all of which various grades of stupor and acute clouding of consciousness occur. The acute onset in every type carries with it a serious prognosis, and often ends fatally. On the other hand, the acute symptoms may occasionally subside and the disease may thereafter progress in the ordinary chronic form. The pathological change in these acute cases consists of an extensive and active meningo-encephalitis. It is suggested by the authors that the acute clinical symptoms may have arisen because of the pronounced vascular reaction and intense perivascular infiltration. Immediate treatment is imperative. Injections of "Tryparsamide", should precede the institution of fever therapy, which is generally more efficacious if given after the "Tryparsamide". Soluble preparations of mercury or daily intramuscular injections of red iodide of mercury have been recommended as preliminary treatment.

Convulsion Therapy by Ammonium Chloride.

THE search for the ideal therapeutic convulsant goes on. Injections of ammonium chloride to produce convulsions were first introduced by Bertolani, according to E. Cunningham Dax (*The Journal of Mental Science*, July, 1940), who analyses the clinical reactions of the drug when it was used in the treatment of twenty-four psychotic patients. Its effects, he claims, are milder than those produced by the well-known convulsants "Cardiazol" and "Azoman"; and patients who have had experience of all these drugs are said to prefer the injections of ammonium chloride. As similar injections of sodium chloride produce no

convulsions while injections of ammonium bromide have been found to produce convulsions, the author believes that the ammonia is the convulsive radicle. Ten cubic centimetres of a 5% solution of ammonium chloride are regarded as the average convulsant dose. The drug is given intravenously and as rapidly as possible. The reaction appears to follow a set pattern: (*a*) In the preliminary stage the patient shows apprehension, may become red in the face and clutch the bed-clothes. (*b*) In ten to twenty seconds after the injection there is hyperpnœa with pupillary dilatation and deviation of the head and eyes. The pupils are inactive to light and the corneal reflex is lost. Blotching may occur about the neck and chest. The face may be congested, and there sometimes develops a circumoral pallor. (*c*) From ten to twenty seconds later the head deviation is replaced by convulsive movements, which, although varying in degree, never approach the severity of those produced by "Cardiazol". There is no cyanosis, and incontinence does not occur. Consciousness may be regained before the muscular movements have ceased. (*d*) The position in which the convulsion ends is maintained for a few seconds, after which the patient's conduct is variable. There is generally a transient stage of confusion, but some patients awaken quite rational. Nausea is rare and headaches are infrequent. Ten minutes after the convulsion the patient may be allowed to sit up and have a meal. The injections are given twice a week and the indications for the treatment are the same as those for "Cardiazol" or "Azoman". Although the results of ammonium chloride therapy are not so good as those obtained by the use of "Cardiazol", the author believes that it is useful in selected cases, particularly when the patients are elderly and bed-ridden. It is certainly less unpleasant, and the risk of its producing fractures or dislocations is negligible.

Late Effects of Subdural Hæmorrhage.

A STUDY of fifty-four cases of subdural hæmorrhage is presented by Eric A. Linell (*The Lancet*, October 26, 1940). Though free hæmorrhage into the subdural space is a common and frequently fatal incidental finding in cases of head injury, chronic hæmatoma may follow a minor injury with no serious intracranial damage, and there may be no history of trauma. In such a case mental deterioration may follow. Cases are quoted illustrating the early and late onset of mental symptoms resulting from subdural hæmorrhage of traumatic origin. Headache and pain were followed by irritability, alternating with somnolence and then confusion. In one of these cases the hæmatoma was successfully removed nine years after the original accident. The author believes that infection of the hæmatoma is a rare complication in civil life and is almost invariably fatal.

Alcohol Injection of the Gasserian Ganglion for Migrainous Neuralgia.

WILFRED HARRIS (*The Lancet*, October 19, 1940) distinguishes between migrainous neuralgia and migraine by the absence of premonitory symptoms and the rarity of subsequent vomiting or nausea. He describes the typically unilateral headache which begins suddenly with a pain of such violence that the patient may dash his head against the wall. There is an absence of "trigger zones" characteristic of trigeminal neuralgia. In discussing the pathology of the condition the author claims that there is a strong presumptive evidence of an intracranial vasomotor disturbance. For this reason he treats it by alcoholic injection of the Gasserian ganglion, and illustrates his method by quoting the case histories of five patients. Of the 29 patients treated by this method, 19, it is claimed, have been completely relieved and five others much improved.

Cerebro-Spinal Fever Treated with Sulphapyridine.

AN epidemic of cerebro-spinal fever in a Royal Air Force hospital led Denis Williams and Denis Brinton (*The Lancet*, October 19, 1940) to investigate methods of treatment, and forty-five consecutive cases were used to determine the best method of administering sulphapyridine ("M & B 693"). All patients recovered and all were fully examined within twenty-four hours of their discharge from hospital when no cranial nerve palsies were visible. The authors discovered that only by giving large initial doses of the drug could the concentration of the sulphapyridine in the blood and cerebro-spinal fluid be raised to an adequate level. It was tolerated and the likelihood of toxic symptoms was minimized. Two grains were given on admission to hospital, followed by two grains every two hours for two doses and then one grain every four hours. On the second day one grain was given every four hours, on the third day every six hours, and thereafter at eight-hourly intervals until 30 grains had been given, by which time all signs of meningeal irritation had usually disappeared. The concentration of sulphapyridine in the cerebro-spinal fluid was higher in a group treated without thecal drainage than in those in which lumbar puncture was performed. Lumbar puncture to relieve intracranial pressure and to give symptomatic relief is, in the author's view, unnecessary, because with sulphapyridine the period of grave illness is so much shortened.

Ecological Studies of Mental Disorders: Their Significance for Mental Hygiene.

H. WARREN DUNHAM (*Mental Hygiene*, April, 1940) has studied the distribution of the various types of mental disorder in the urban environment. The material included all persons from Chicago who between 1922 and 1935 were admitted for the first time to mental hospitals. The author found that the highest rates of the schizophrenic group were concentrated in the most disorganized areas of the city, which suggests that social factors are more significant in the schizophrenic psychosis than in the manic-depressive disorder. He also found that high rates of schizophrenia occur consistently in the ethnic groups that are the minority population groups in various communities of the city—that is, for example, where native-born white stock predominated, schizophrenia was more prevalent in Negro families and *vice versa*. The distribution of paranoid schizophrenic types was highest in the rooming house areas of the city, while the highest rate for the catatonic types was among the foreign-born and poorer classes. These facts indicate that different types of mental disorder are related to different types of community; and they serve to emphasize the importance of studying the cultural organization of the various ethnic groups in the urban population and to provide the essential sociological basis for the establishment of a mental hygiene programme which the now rapid increase in mental disorder has rendered of paramount importance.

The Long-Term Prisoner.

IMPRISONMENT, the currently accepted method of treating offenders, is, according to Marvin Sukov (*The American Journal of Psychiatry*, May, 1940) not successful. Recidivism is a common occurrence; and some help is needed towards the achievement of a satisfactory extramural adjustment. In the case of long-term prisoners, incarceration has been found to produce progressive social severance. Prisoners subjected to a life sentence of twenty or more years felt that though punishment was justified, the sentence was too severe. Their imprisonment had little effect in the intellectual sphere; and after many years' imprisonment the subjects were found friendly and without antagonism to society. While there is an expressed desire for freedom the writer is of the opinion that in the case of the long-term prisoner life outside the prison would not be possible without some definite help to accomplish a stable adjustment.

Alzheimer's Disease.

SIX cases of Alzheimer's disease are recorded by W. H. McMenemey (*Journal of Neurology and Psychiatry*, July, 1940) and a plea is made for a wider conception of this disease. Full clinical details and histological findings are presented and compared with those already in the literature. The author considers it inexpedient to regard this disorder as a presenile dementia. Attention is drawn to certain familial factors in Alzheimer's disease which might lend weight to its being an abiotrophy. The present author suggests that there are two factors involved—endogenous and exogenous. The former is most marked in the familial cases; and the latter is evident in certain examples of a subacute toxic-infective psychosis with dementia which at autopsy present the characteristic histological cerebral changes. Many gradations appear between the endogenous and exogenous types; and many are doubtless still overlooked or wrongly diagnosed.

Fractures of the Vertebral Bodies.

A DISCUSSION of the cause of fracture of the vertebral bodies, particularly in the mid-thoracic region, is thought pertinent by A. M. Rechtman (*The American Journal of Psychiatry*, May, 1940), who has observed such injuries follow therapeutic convulsions in psychotic patients. He considers that the degree of flexion of the spine during a convulsion occurs through an arc sufficiently small to make the mid-dorsal spine the recipient of its greatest effects. The number of convulsions has no relation to the number of vertebral bodies fractured; but the extent of the injury is dependent on the intensity of the muscular contractions and the strength of the spine to withstand the shock. No mechanical fixation can prevent this type of injury. Rupture of the *nucleus pulposus* with protrusion into the neural canal is most unusual. Prophylaxis is possible by the use of spinal anæsthesia prior to the convulsion. Fixation in a plaster jacket is the treatment recommended.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

XXX.

ALCOHOL AND TRAFFIC ACCIDENTS.

A COMMITTEE of the British Medical Association in 1927 decided "that the word 'drunk' should always be taken to mean that the person concerned was so much under the influence of alcohol as to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation on which he was engaged at the material time".

The onus of responsibility for traffic accidents falls most heavily on the driver of a motor vehicle, as the speed of which such a vehicle is capable demands for safe driving a controlled state of mind and competent neuro-muscular coordination. Responsibility, particularly in regard to neuro-muscular control, is demanded of cyclists, and to a less extent of drivers of horse-drawn vehicles and pedestrians. Even so, Marpother has stated that 22% of accidents in the United States of America in which pedestrians are involved are the result of intoxication on the part of the pedestrian.

Alcohol acts predominantly on the central nervous system, and in proportion to the amount consumed progressively impairs the functions of this system.

The British Medical Association committee pointed out that "the first effect of alcohol is on the higher centres and is subjective, and, even if no objective symptoms occur, the subjective effect may be sufficient to make it unsafe for an individual to be in a responsible position".

Most people are subject to reaction tendencies against feelings of inferiority. A symptom of this in the motor-car driver is an intolerance of being behind others and a determination to pass other vehicles on the road. The urge to travel at high speed may also originate from an instinctive craving for power, violent emotion or euphoria. Alcohol, by reducing the inhibition of the highest centres, tends to release these motivations towards speeding.

Whately Smith measured the influence of alcohol on emotion. He found that the psycho-galvanic reflex was diminished in magnitude and variability, that the proportion of inner association to stimulus words was smaller, but that the proportion of highly toned reactions increased at the expense of the moderately toned. He concluded that these experimental results indicated that "under the influence of alcohol a person is on the whole more inert in that many stimuli produce a smaller effect in him than in the normal person, but that if the stimuli once surpass a certain value the result in behaviour is extreme". McDougall found that one of the earliest signs of the effect of alcohol was a striking decrease in the rate of apparent alternations of direction of rotation in a "windmill" illusion. He also found that memory of relationship between listed words was diminished. The laboratory results obtained by Smith and McDougall are reproduced in everyday life in motorists by a tendency to ignore the courtesies of the road and traffic indications, and on the other hand to take undue risks.

At the reflex level the action of alcohol again leads to depression of function, though its action at the higher levels creates at the same time a conviction of increased ability. In some subjects with introverted personalities and abnormally developed inhibitions, the earliest effects of alcohol may release more efficient reflex responses; but this is unusual. Reaction times, the blinking reflex and the latent period of the knee jerk are prolonged, the rate of typewriting is slower. With a dotting machine more errors are made. Habitual movements, such as are involved in dressing and undressing, lighting and putting out a cigarette and picking up objects from the floor, may be fumbled. Equilibrium in posture and movement may be less stable than normally. Articulation may be slurred and the pronunciation of difficult words and phrases may be poorly executed. Handwriting may show signs of incoordination.

In a very ingenious apparatus used by Miles an indicating needle was connected to a steering wheel and the subject had to keep the needle on a moving mark. After he had drunk one and a half pints of very light beer, errors increased by 11%.

With larger amounts of alcohol the pupil reaction becomes sluggish to light. This "tonic reaction" is a normal feature in some persons. With still larger quantities of alcohol mentality becomes more infantile and confused, conduct more grossly emotional and action more incoordinated, until the stages of coma or even death from respiratory paralysis may eventuate.

Although in general the stages of depression of the central nervous system follow in order from the most specialized to the most automatic, yet there are wide individual variations. One young man whom I used to know could carry on a highly intellectual discussion after his legs had failed to support him. An alcoholic amnesia for an hour or more, during which time the subject has been mentally and physically active, is a possibility. A characteristic of this amnesia is a gradual fading of consciousness, with patches of memory but an abrupt return to full consciousness. The return to consciousness from alcoholic coma is slow and patchy.

The pharamcological effects of the same amount of alcohol are extremely variable in different persons, and even in the same person in varying circumstances. Taken on an empty stomach, alcohol is fully absorbed and appears in maximum concentration in the blood in a few minutes up to two hours, on an average in about three-quarters of an hour. A carbohydrate and protein meal has little effect on the blood alcohol curve. When alcohol is taken after or before a full meal containing fats, the peak concentration of alcohol in the blood is reduced and is delayed. Widmark states that up to 17% of ingested alcohol may remain unabsorbed after a full meal. Mellanby states that malt liquors and diluted spirits are absorbed more slowly than concentrated spirits. Kionka's experiments did not confirm this. Unmatured wines and spirits are more toxic than matured forms. The rate of ingestion is important. Body weight has a decided influence. Habituation is also important; such tolerance is ascribed by Mellanby to slower absorption, and by Pringsheimschen and Kionka to increased rate of oxidation. Ketosis may raise the apparent blood alcohol content by 30 milligrammes *per centum*, rarely more.

Widmark has suggested a formula by means of which the amount of alcohol consumed can be correlated with the blood alcohol content at certain times. Widmark's original publications are not available; but the formula, quoted by Smith and Glaister in "Recent Advances in Forensic Medicine", does not conform to observed data.

Goldhahn quotes Widmark's figures for the relationship of observed blood alcohol content and the probability of intoxication, as follows (the percentages are percentages of persons):

70 mgm.%	of blood alcohol,	intoxication nil
90 mgm.%	of blood alcohol,	intoxication in 10%
110 mgm.%	of blood alcohol,	intoxication in 30%
130 mgm.%	of blood alcohol,	intoxication in 50%
150 mgm.%	of blood alcohol,	intoxication in 70%
170 mgm.%	of blood alcohol,	intoxication in 80%
210 mgm.%	of blood alcohol,	intoxication in 92%
270 mgm.%	of blood alcohol,	intoxication in 100%

Andersen rules that when an examination shows an alcohol concentration in the blood of 240 milligrammes *per centum*, it is certain that the person was under the influence of alcohol at the time. If the alcohol concentration of the blood is between 150 and 240 milligrammes *per centum*, this finding will confirm a clinical diagnosis that some degree of drunkenness existed.

The clinical diagnosis of drunkenness after accidents is all important and is frequently contentious. The British Medical Association committee emphasized the fact that there is no single test taken by itself which could justify a medical practitioner in deciding that a person is drunk. Consideration has to be given to a number of observations, such as general demeanour, congestion of conjunctivæ, state of the tongue, smell of the breath, character of the speech, manner of walking and turning sharply, sitting down and rising, picking up a pencil or coin from the floor, memory of incidents within the past few hours and estimation of their time and intervals, reaction of the pupils, character of the breathing, especially in regard to hiccup.

Baldie places considerable importance on such habitual performances as the lighting and putting out of a cigarette and the movements of dressing and undressing, following a leader or a crooked line, writing to copy and dictation, copying a diagram, picking a piece of "Elastoplast" from the floor, reading tests, calculations and turning up an index reference.

Due consideration must be given to the possible existence of the following conditions: first and foremost, shock from the accident; then hypoglycæmia, carbon monoxide poisoning and organic nervous or metabolic diseases.

When a medical practitioner is called in by the police to examine a suspected person, the English rule that before examination the patient must be given the right to call a doctor on his own behalf should always be observed.

A select committee of the House of Lords presented a special report on the prevention of road accidents in May,

1940. The committee strongly recommended that all available means of propaganda should be employed to warn people of the dangers of even a small quantity of alcohol for many drivers; that full penalties should be enforced uniformly when the court had found that the driver was under the influence of drink when an accident occurred; and that the finding of a concentration of 150 milligrammes *per centum* of alcohol in the blood should be proof that the person was under the influence of drink at the time.

H. K. FRY, D.S.O., B.Sc., M.D., D.P.H., F.R.A.C.P.,
Honorary Physician, Adelaide Hospital, Official Visitor, Parkside Mental Hospital, Adelaide.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, East Melbourne, on November 13, 1940, DR. H. C. COLVILLE, the President, in the chair.

Chemical Warfare.

DR. IVAN MAXWELL, a member of the Chemical Defence Board, delivered an address on "Chemical Warfare" (see page 97).

DR. W. OSTERMEYER said that the address was comprehensive and lucid, making an enormous impression on him. It had been a privilege to be present and to learn so much from a lecturer with a masterly mind and personality.

DR. G. A. PENINGTON asked Dr. Maxwell what was the probable life of the service kind of canister for protection from noxious gases. He knew that they must not be used unnecessarily, but for training purposes they had to be used at times and he had felt at a loss to know when they should be replaced.

PROFESSOR MARSHALL ALLAN said that he had had an early experience of chemical warfare outside Ypres in April, 1915. On that occasion the soldiers had no protection, but used to soak a sock or handkerchief in a solution of sodium bicarbonate and hold it over the mouth and nose. He thanked Dr. Maxwell sincerely for his informative address and masterly summary of the great advances in their knowledge and technique made since 1915.

DR. C. L. PARK asked Dr. Maxwell to supply further information as to how an untutored civilian was to know immediately that the air had been contaminated by a poison gas.

The President, before calling on Dr. Maxwell to answer the questions, referred feelingly to the sense of gratitude he and those present had to Dr. Maxwell, who had obviously mastered the subject and gone to a great deal of trouble to carry out the wishes of the members of the Science Subcommittee who had arranged the address. In selecting Dr. Maxwell for the task they had had every confidence that the subject would be presented in the best possible manner for the information and instruction of the members of the Branch. He hoped that the address would be published in THE MEDICAL JOURNAL OF AUSTRALIA and that the members would read it carefully and preserve it for future reference.

Dr. Maxwell thanked the President and members present for the cordial way in which they had received his address. He had given addresses on the same subject a number of times recently, but they had not been published. He found it very difficult to answer Dr. Penington's questions. They had several gases to consider, such as mustard gas, lewisite, phosgene and arsine, and the life of the respirator varied for each gas. Moreover, the materials in the respirator had a certain power of recovery. A definite answer could not be given. Samples should be taken and tested, and the opinion expressed concerning the samples could be applied to the group.

In reply to Dr. Park, Dr. Maxwell expressed the opinion that civilians must learn as much as possible about the characteristic smells of the gases likely to be used in warfare. There were, however, certain chemical detectors, such as the one for arsine, which could be multiplied and widely distributed.

In conclusion, Dr. Maxwell thanked Mr. P. R. Weldon, of the Chemical Defence Board, who had supplied some of the lantern slides used by him that night.

Naval, Military and Air Force.

THE following clinical report from the 2/1 Hospital Ship *Manunda*, Australian Imperial Force, has been received from Major-General R. M. Downes, Director-General of Medical Services. The report was compiled by Major C. H. Osborn, Major C. Sippe, Captain C. P. Manson and Captain W. Freeborn, of the Australian Army Medical Corps, and medical officers on *Manunda*.

"UNFIT FOR ACTIVE SERVICE."

A clinical survey of the soldier patients transported to Australia from the Middle East on the first voyage of the Hospital Ship *Manunda* was conducted by the medical officers of this unit, and the results are set out herewith. It is hoped that such a survey and the conclusions derived therefrom may prove of interest and benefit to officers of the Australian Army Medical Corps who are engaged on medical boards in Australia, and may help them to assess the physical suitability of potential recruits for foreign service. It is not to be regarded in any sense as a reflection upon the efficiency of previous examinations, as we fully realize that in many instances patriotic zeal has overshadowed the truth and important details of past medical history have been deliberately withheld by the examinees.

All the patients carried on this voyage, and they include men from the New Zealand contingent as well as members of the Australian Imperial Force, have been boarded out as unfit for further service in the army abroad.

In view of the high incidence of certain diseases or infirmities amongst this representative group of invalided soldiers, it has been possible to consider several main divisions, and it is interesting to note that the numbers in these provide a very large percentage of the whole. The remainder are a varied assortment, as one would expect with any large body of men, and include the late results of acute disease, accidents *et cetera*. It is not our purpose to analyse this miscellaneous group, as no fruitful conclusion could be drawn therefrom; but it is highly gratifying to note, and reflects praise upon the army medical authorities, that the incidence of tropical diseases and of venereal infection is almost negligible.

The disabilities can be roughly classified into two main groups, as follows: (i) those present prior to enlistment, which have become aggravated by training; (ii) those which originated on active service. In each of the disease classifications the distinction between the above two main groups has been shown, and in this way it is hoped to demonstrate more clearly those men who are likely to break down on foreign service.

This study has been facilitated by the method of admission adopted on this ship, whereby patients with diseases of a similar type have been segregated into particular wards and placed under the care of individual officers. Investigations that had not already been carried out in base hospitals were completed on the ship, and when indicated appropriate medical or surgical treatment was instituted.

Diseases of or Injury to the Nervous System or Mind.

Anxiety States.

The term anxiety state means that perpetual *angor animi* developed by people of unstable psychical make-up when they are subjected to an unusual stress or strain. There were 34 such cases, and they fall naturally into three subgroups, as follows: (i) anxiety states developed after enlistment without any previous history of "nerves" or "nervous breakdown"; (ii) anxiety states associated with a previous history of mental breakdown; (iii) anxiety states with visceral fixation—(*a*) functional dyspepsia, (*b*) cardiac neurosis. Table I shows the distribution of such sub-groups.

TABLE I.

Sub-group.	Average Age. (Years.)	Months' Service Before Breakdown.	Remarks.	Number of Patients.	Average. (Percentage.)
(i)	20·0	4·5	50% suicidal.	8	14·0
(ii)	35·5	3·0	None suicidal.	14	50·0
(iii) (*a*)	31·0	6·0	Only one without previous dyspepsia.	6	20·6
(iii) (*b*)	35·0	10·0	All with a previous history of "turns".	8	15·4

Suicidal tendencies were evident in only those cases belonging to the first sub-group, and at least half the patients of this sub-group attempted self-destruction. This was the first indication of mental derangement in several cases.

Careful inquiry into the past histories of these patients shows that they were capable of "carrying on" in civil life in spite of minor breakdowns; but the strain of military life on active service quickly revealed their mental instability. A large number might have been excluded from the army if a truthful history had been given in the first place; but, failing this, there is no certain method whereby these potential invalids may be detected. The restless, underweight, obviously neurotic type with trembling clammy hands is always a candidate for this group.

Epilepsy.

There were eight cases of epilepsy, none being severe. The average age was thirty-three years, and all the patients were physically very sound and well up to an average standard of mental ability. Four had lesions of the idiopathic type, three considered a previous head injury to be the cause of the seizures, and only one had developed the disease after enlistment.

Post-Concussional Headaches.

There were seven cases of post-concussional headache; all patients had the symptoms so severely that they were useless for active service. All had definite histories of severe head injuries, with periods of unconsciousness at the time of the accident ranging from a few hours to several days. In many instances scalp scars could be seen or irregularities felt in the bones of the skull. The average age was thirty-three years, and without exception the onset of severe attacks began during the first week of serious training. All the patients were of poor physique, and with two exceptions much below standard in mental acuity.

Mental Aberrations.

There were twelve cases of mental aberration; five of the patients had schizophrenia (four of them had had treatment for mental breakdown prior to enlistment), five had the manic-depressive syndrome and no previous history was obtainable, and three had congenital mental deficiency and were physically sound, but obviously "high-grade aments". The condition of the last three could be diagnosed from a few moments' questioning and conversation, and all three could neither read nor write. The average age of the patients with schizophrenia was twenty-four years, and the average age of those with the manic-depressive syndrome was thirty-five years. These were of poor physical type and had been admitted to hospital after an average of only four months' service.

Discussion.

These facts show clearly than an accurate and truthful history would have prevented many of these men from entering the army: the exceptions are one epileptic and the patients with acute mental diseases who first manifested symptoms after enlistment. The obvious mental deficients should never have appeared on service at all.

The post-concussive headache or syndrome deserves special consideration, for patients in this group could stand no training at all. We were firmly convinced by a study of these and previous similar cases on the ship that no recruit with a history of severe concussion or head injury should be accepted for active service. The presence of more than a small scar on the scalp or any non-physiological bony irregularity in the skull should be regarded with suspicion.

Diseases of the Respiratory System.

Diseases of the respiratory system are divided into (i) non-tuberculous disease (asthma) and (ii) tuberculous disease.

Non-Tuberculous Disease.

TABLE II.

Asthma, Bronchitis and Bronchiectasis (41 Cases).

Percentage of Total.	Ages.		Number Showing Symptoms Prior to Enlistment.	Number Showing Symptoms Only after Enlistment.
	Years.	Number of Patients.		
12·3	20 to 29 30 to 39 40 and more	14 15 12	33 (80%)	8 (20%)

This comparatively large group, comprising 12·3% of the total number, is of importance, especially as 80% of the men had a previous history extending over a number of years. In some instances the symptoms had commenced in childhood. The high percentage (80) with a previous history strongly supports the instructions given to examiners that a previous history of asthma should exclude a recruit.

In the main, symptoms commencing after enlistment had followed an acute respiratory infection — probably the unmasking of a latent allergic state. Frequently climatic influences were cited as precipitating or aggravating factors; but undoubtedly the nervous element was, in many instances, of paramount importance.

It is interesting to note that 29·2% of the group were aged over forty years, whereas of the total sick 16·25% were of this age group.

Any soldier who has suffered from asthma or a long-standing cough, or in whom rhonchi can be detected in the lungs in the absence of a definitely acute infection, should be excluded. Even patients with acute infections should not be beyond suspicion. In this connexion the importance of examining the lung bases after the patient has been coughing is often not sufficiently appreciated.

Pulmonary Tuberculosis.

The classification of a group of 17 patients with pulmonary tuberculosis has been made along different lines, as 11 patients without symptoms have been returned on account of lesions discovered in the X-ray photographs made prior to embarkation. Five soldiers had "reported sick", complaining of symptoms including cough, hæmoptysis, loss of weight and chest pain. There was no record in the available papers of the result of the routine X-ray examinations. In one other case no routine X-ray examination had been made.

In all cases the diagnostic films suggested a chronic lesion of more than a few months' duration. In three cases at least there were signs of recent activity as well. Two circumstances could have led to the inclusion of these men in the Australian Imperial Force: (i) the miniature films were deficient, (ii) in the early stages radiologically healed lesions were accepted.

This aspect needs further consideration and a reexamination of the miniature films. The type of lesion seen strongly suggests the advisability of not accepting even patients with apparently healed parenchymatous lesions.

At least some of the 11 men who had not developed symptoms, viewed in the light of the other group, would have broken down. This provides further proof, if such proof is needed, of the usefulness of routine radiological examination of the lungs. The only doubt which is raised is as to whether some of the active lesions had been overlooked on the miniature films.

Cardio-Vascular System.

The functional cardiac states are discussed under the headings of nervous disorders. There were one case of long-standing valvular disease, four cases of recent valvular disease, and one case of arteriosclerosis.

The small number of cases of established disease is noteworthy—one of valvular disease and one of arteriosclerosis. This should be taken to indicate a very efficient physical examination prior to enlistment. The four recent cases have followed attacks of rheumatic fever occurring on service. One man is still in bed with an active cardiac lesion. Another had suffered from several previous attacks of acute rheumatism, and the cardiac lesion may have been associated with these earlier attacks. It is possible that some men with chronic valvular disease may still be carrying on and may even continue through years of strenuous service.

Diabetes Mellitus.

Diabetes mellitus is of importance because of the severity of at least two of the five cases. The urine of these men was not sugar-free when they received a limited diet and 70 units of insulin per day. A limited supply prohibited a further increase in the dosage. No previous history was ascertainable in any instance.

There is always the possibility that the urine of a diabetic patient may be made sugar-free at the time of examination by the use of insulin or that a false specimen of urine may be presented. The latter possibility can be coped with by close attention to urine collection and examination; the former would be very difficult to combat.

Peptic Ulcer.

The high proportion of patients suffering from peptic ulcer demands careful consideration, and an analysis of the figures shown in Table III reveals some interesting facts.

TABLE III.

Number of Cases.	Duodenal Site.	Confirmed by X Rays or Operation.	Average Age in Years.	Average Duration of Symptoms in Years.	Wearing of One or More Dentures.	Average Weight.
42 (13·1%)	34 (81·0%)	34 (81·0%)	34·8	5·8	36 (85·7%)	9 stone 12 pounds

Probably the most useful information concerned the high proportion (88%) of these invalids in whom symptoms of dyspepsia had been present prior to enlistment, and no fewer than nine had undergone operations for ulcers. In only five instances were the patients convinced that the symptoms had originated on active service. This suggests that active service conditions are more potent in precipitating a relapse than in producing the disease *de novo*, and confirms the accepted view that patients with peptic ulcers should exercise lifelong care with regard to their diet and living conditions.

Positive radiological findings had been recorded in 31 cases; three patients who had been subjected to a previous operation for ruptured ulcer were not thus examined.

The average age of the group was 36·4 years, compared with an average in all cases of 32·2 years.

Manson-Bahr is very emphatic on this question; he states, in speaking of suitability for tropical conditions, that "subjects of nervous dyspepsia, hyperchlorhydria or severe gastric disturbances should not go abroad" (P. H. Manson-Bahr, "Tropical Diseases", 1940, page 4).

In addition to the nine patients operated upon for ulcers, 17 had been subjected to appendicectomy. In a certain proportion this operation had been performed in an endeavour to cure the dyspepsia. The presence, therefore, of upper or lower abdominal scars should lead to a very careful questioning with regard to dyspeptic symptoms.

On embarkation only 19 patients were dentally fit, and 36 (86%) wore at least one denture. As regards weight, 18 weighed under ten stone, the average weight being nine stone twelve pounds.

Conclusions.

1. Any recruit with proved peptic ulcer, active or quiescent, should unequivocably be rejected for service abroad.
2. If there is a dyspeptic history of more than a temporary nature, it would be advisable (*a*) to reject the recruit; (*b*) to submit him to the appropriate laboratory investigations and review the position in the light of these findings.
3. The presence of one or more abdominal scars should evoke careful questioning and due consideration of all the possibilities involved. An operation for chronic appendicitis may indicate a long-standing dyspepsia, and an upper abdominal scar may indicate a ruptured ulcer.

Backache.

The incidence of painful back as a cause of invalidism is surprisingly high, and a careful clinical and radiological review of these cases reveals several interesting points. In all cases pain was complained of in the lower part of the back, usually on each side of the mid-line, but sometimes on only one side, and in many instances pain is referred down one or the other thigh. The anatomical site of the pain only occasionally gives a clue to the source, on account of the innumerable joints, muscles and aponeuroses in this area, all of which may be affected by strain. Furthermore, congenital abnormalities affecting the bones and joints in this region appear to make the individual more susceptible to injury.

As age advances, so does the back appear to become more vulnerable to strain, and in this series the age factor is quite noticeable; the average age of the group is 38·6 years, and there are only six patients under thirty-five years of age. Chronic backache is a most disabling complaint, and the relative absence of physical signs frequently results in the labelling of such soldiers as malingerers. True, there may be some; but the soldier whose back aches constantly during a long march or tour of duty is a sufferer indeed and cannot hope to be efficient. The more acute forms of myofascial and ligamentous strains are immediately noticeable by the obvious sharp stabs of pain and the "cast position" the patients assume — the so-called "sciatic scoliosis". Repeated attacks of this nature make the sufferer completely unfit for active service.

When X-ray examination reveals arthritic changes in the lumbar part of the spine, lumbo-sacral region or sacro-iliac joints, and when the clinical findings are consistent, such lesions have been regarded as the source of pain. In many of the cases the symptoms are in a quiescent phase after a long period at the convalescent depot; but it is fairly characteristic of these lesions for the symptoms to wax and wane, and many of the soldiers had sincerely tried to resume training with their units, only to find that the painful symptoms recurred. Investigations for sources of focal sepsis were carried out, but apart from dental sepsis, all of which had been already dealt with, nothing of note was detected.

Low Back Pain.

Low back pain was classified as follows:

Number of cases: 25, or 8% of invalids.
Average age: 38·6 years; average age of all invalids, 32·2 years.
Bone and joint changes (shown radiologically): 11 cases.
Myofascial strain or lumbar fibrositis: 14 cases.
Long history of low back pain: 19 cases (arthritis 10, fibrositis 9).
Symptoms first noticed during training: arthritis 1 case, fibrositis 5 cases (total 6 cases).

Over three-quarters of the patients suffering from low back pain admitted previous pain following injuries in civil life; but as there were no satisfactory clinical tests to detect potential sufferers amongst recruits, a careful inquiry into the past history of back strain should be made, particularly in men aged over thirty-five years.

Foot Disabilities.

The special attention paid to examination of the feet of recruits has avoided any large wastage owing to foot disabilities, and only eight soldiers are being invalided home on this account. The nature of these is shown in Table IV.

TABLE IV.

Abnormal Condition.	Number of Cases.
Hallux valgus	2
Hallux rigidus	1
Old fractures of foot or ankle, with deformity	2
Painful flat feet	2
Congenital talipes	1

With the exception of the two patients with painful flat feet, all the remaining patients showed obvious deformity at the time of enlistment. In most instances symptoms did not intervene until military boots were worn, or long marching became necessary. It is probably unwise to accept any recruits for foreign service with deformities of the feet in spite of modern mechanization.

Lesions of the Knee Joint.

There were 12 patients invalided home on account of lesions affecting the knee joints, and the average age of the group was 33·5 years. In all cases, except one of pronounced congenital *genu varum*, the joints had caused no disability to the men in the pursuance of their civil occupations; but with the strain of military training aching pain was experienced, sufficient to disable them.

Several patients showed definite evidence of osteoarthritis, and all of these had a history of previous injury (see Table V).

TABLE V.

Lesion.	Number of Cases.
Osteoarthritis	7
Chronic traumatic synovitis	2
Internal derangement	1
Recurrent dislocation of patella	1
Congenital *genu varum*	1

A history of trauma to the knee joint, including injuries to the internal meniscus, should demand careful survey of the joint.

Conclusions.

Our investigation of these invalids confirms the instructions already issued by the Director-General of Medical Services regarding the standards of physical fitness for recruits. Although we appreciate the difficulties that confront the examining medical officer, particularly in a rush period of enlistment, it must be realized that the great expense alone of sending one unfit man overseas should outweigh all personal desire to do that man a so-called "good turn" by passing him as "fit for service".

Remember that hospitals near the war zone must not have beds occupied by soldiers who have "cracked up" during the training period, and this can be obviated to a large extent by careful preselection.

A review of these groups reveals a low incidence of diseases with detectable physical signs. The larger groups consist of those conditions which cause symptoms rather than physical signs, such as chronic backache, peptic ulcer and nervous diseases. Another large group consists of asthma and bronchitis, in which diseases there may be a complete absence of physical signs during a remission. The inference is that, although the physical examination of recruits has been of a high standard, sufficient emphasis has not been laid on a careful clinical history.

Study of the Questionnaire.

A study of the D1 forms suggests an adequate explanation for this defect. In nearly all cases the answers to the *questionnaire* had been filled in either by the soldier or by a clerk who obviously would not know the type of subsidiary questions to pursue. The importance of this will be appreciated when it is realized that, of the number of invalids classified (208), 74% gave a long history of preexisting disease aggravated by service. (This excludes pulmonary tuberculosis.) In spite of the tendency of a keen recruit to withhold this information, close questioning can frequently overcome this difficulty.

It is at times possible to suspect asthma from radiological appearances in the chest, and questioning in such cases will repay the effort. In this group the physical appearance of the chest is frequently of more importance than the degree of expansion, which in many cases is a trick movement.

Furthermore, the important questions concerning digestive disturbances have been omitted. These should be added as soon as possible, to act as a reminder to the examiner. It is also seen that insufficient regard has been paid to upper abdominal scars—not so much as to their firmness, but as to what underlying pathological lesions they indicate.

Just as in clinical medicine, more time could with advantage be spent on a study of the past history, even at the expense of slowing down the rate of examinations. In some cases more time is spent over a doubtful blood pressure than is given to a study of past history. A soldier who has suffered severely from asthma or a peptic ulcer is more likely to break down than one with a slightly raised blood pressure.

Pulmonary Tuberculosis.

The comparatively large number of men suffering from pulmonary tuberculosis (5, or 1·5% of the total) who had apparently been passed as fit seems to need investigation. The miniature films need rechecking, although they may represent radiologically healed lesions in patients who were at first accepted.

The age distribution in the total is of interest:

20 to 29 years	118 (36·9)
30 to 39 years	140 (43·8)
40 to 49 years	54 (16·8)
50 to 60 years	8 (2·5)

The average age is 32·2 years. Thus 62, or 19·3%, are above the maximum age. It is unlikely that nearly 20% of the Australian Imperial Force are above forty years of age.

A more careful consideration of age is suggested, and in doubtful cases some check could be made by the production of birth certificates. In cases in which the physical age appears greater than the stated age, even in the absence of detectable abnormalities, rejection is the wisest plan. This is the method at present advised, and the cases cited support this advice. Unfavourable conditions are frequently at their worst on active service. It is difficult to avoid excessive fatigue, irregular and insufficient sleep, with variable and often imperfect diet. The soldier is inclined to smoke more than in civil life, and any incidental infection—for example, influenza, sand-fly fever, dengue fever *et cetera*—would be an added strain on his reserve.

Nervous symptoms have been of frequent occurrence in all groups, including the organic states. The stress and strain of war service, even during the training period, is far greater than that of civil life, in which many of these men are able to carry on and lead useful lives.

The main conclusion is that a more painstaking use of the *questionnaire* will at least reveal some of the preexisting diseases and prevent the acceptance of recruits a large number of whom cannot possibly give good service. The total number of troops returned on medical grounds is comparatively small, and is in itself a tribute to the judicious care in the early medical examinations, and to the experienced wisdom of the instructions issued by the Director-General of Medical Services. The imposition of an unduly high physical standard is bound to result in individual hardship and waste of man power, and no man who is reasonably fit should be deprived of the opportunity of serving his country. On this account it is desirable to emphasize again those factors of age, congenital or acquired defects and constitutional tendencies which are liable to render a man unfit in the light of experience gained by a clinical study of those soldiers who have already broken down under the strain of active service in this war.

Acknowledgements.

Our combined thanks are due to Lieutenant-Colonel J. R. M. Beith, Commanding Officer of the 2/1 Hospital Ship, who has provided us with every facility to conduct this investigation, and to Major-General Downes, Director-General of Medical Services, for permission to publish it.

Post-Graduate Work.

COURSE IN SYDNEY FOR MASTER OF SURGERY EXAMINATION.

THE New South Wales Post-Graduate Committee in Medicine announces that a course suitable for candidates for Part II of the examination for the degree of Master of Surgery has been arranged to be held from March 3 to May 16, 1941, subject to a minimum of four applications being received. There will be a recess from April 10 to 21.

The fee is ten guineas for the course, which will be held in the afternoons only at the following hospitals: Prince Henry Hospital, Royal Prince Alfred Hospital, Saint Vincent's Hospital and Sydney Hospital.

Copies of the tentative programme are available from the Secretary, New South Wales Post-Graduate Committee in Medicine, the Prince Henry Hospital, Little Bay, to whom applications for registration should be made as soon as possible.

Another course for the M.S., Part II, will be held from September 22 to November 28, 1941, subject to a minimum of four applications being received.

Correspondence.

STATE SOCIAL SERVICES: PAYMENT OF DOCTORS.

SIR: It is stated that the scheme of payment of doctors rendering attention to the poorest members of our community, under the State Social Services Department, was drawn up by the officials of that department and the British Medical Association.

If so, I wonder if the British Medical Association could do anything to rectify the curious situation by which the State pays the doctor if a patient has a miscarriage, but confinements must be paid for by the patients themselves. This means, of course, that the doctor gets nothing at all, especially in the country, where the bonus does not pay nursing home expenses. These mothers, drawn from the poorest homes in the community, are often in bad health and give a great deal of work and anxiety. Surely the State should see that the over-worked country doctor gets some fee for all his trouble.

Recently a patient under this scheme threatened to abort, but she was nursed along and produced a seven months' infant, which is now doing well. If she had aborted I could

have collected a fee, but as it is, I get nothing except the extra worry and trouble of a premature infant under my care.

Many of these women are honest and do without a doctor, and suffer needlessly because they do not wish to incur a debt which they cannot pay.

I hope that in the coming year, both for the sake of the mothers and the doctors, the Social Services Department can rectify this anomaly.

Yours, etc.,

Armidale, New South Wales, January 7, 1941. E. KENT HUGHES.

CRIME AND ITS TREATMENT.

SIR: Twenty-five years ago Dr. E. P. Dark, M.C., afforded us an example of noble effort for social betterment. During the last few years he has stressed the privilege and the duty of the medical profession to study and to alleviate the ills not only of the individual body, but also of the body politic.

He now puts us deeper in his debt by a humane, thoughtful, lucid and stimulating essay on "Some Medical Aspects of Crime" (THE MEDICAL JOURNAL OF AUSTRALIA, January 4, 1941, page 13). Perhaps Dr. Dark will permit three minor criticisms.

1. In his second paragraph he lists three aims of punishment: (*a*) vengeful, (*b*) deterrent, and (*c*) reformative; he might have added a fourth, which indeed he names in the later part of his essay, that is, to protect society.

2. He is scarcely fair to Bentham. Even if the passage quoted is in Jeremy Bentham's "Panopticon", it is not fairly representative. Bentham was extraordinarily kindly and humane; his philosophic utilitarianism and his projects for legal reform have instigated much social amelioration.

3. Dr. Dark shows insufficient appreciation of the determinist theory of rewards and punishments advocated by the late Right Honourable J. M. Robertson ("A Short History of Morals", London, 1920).

Though some may find Dr. Dark's proposed house of reformation too attractive an institution, all may well approve the main argument and content of his essay, and in particular his eloquent indictment of flogging.

Yours, etc.,

131, Macquarie Street, Sydney, January 11, 1941. GUY GRIFFITHS.

SO-CALLED REFLEX ANURIA.

SIR: Dr. Colin Edwards's criticism, in THE MEDICAL JOURNAL OF AUSTRALIA, January 11, 1941, of my article on so-called reflex anuria calls for a reply. I am pleased that Dr. Edwards agrees with me on one very important point, "that the term reflex is almost certainly a misnomer", although I would say, certainly a misnomer, in this type of case. He disagrees, however, with certain of my other observations and conclusions. The signs and symptoms of renal or ureteral stone were apparently absent from three cases mentioned by him as having come under his notice. The only one of the three which I have seen the report of had abdominal pain, vomiting, passed blood-stained mucus *per urethram*, and at operation the kidney pelvis was found distended with blood-stained urine (see report of this case in THE MEDICAL JOURNAL OF AUSTRALIA, September 2, 1939, page 365). If these are not symptoms met with in impacted ureteral stones, then there is abundant evidence of symptoms of stone in many of the other cases reported. The case reported by Barnett and Schlink in THE MEDICAL JOURNAL OF AUSTRALIA, May 13, 1939, had lower abdominal pain and gross hæmaturia before the suppression of urine; and three of my four cases had renal colic and hæmaturia. Dr. Edwards states that there was in his cases "an entire absence of typical colicky pain". It is well known that the pain resulting from calculi blocked in both ureters is not of the severe colicky nature met with where a movable stone is present on one side. Indeed during impaction pain is frequently absent. In reply to Dr. Edwards's inquiry about dilatation of the ureters in my cases. In three of them the upper portions of both ureters, as well as the kidney pelves, were dilated and tensely distended with urine. In the fourth, where only one kidney was examined, the ureter was not dilated because the obstruction was at the uretero-pelvic junction, and one could feel a definite thickening at this point, which I considered at the time to be a hard fibrotic ring. As this was one of my earlier cases, I did not think of a stone being the cause. I incised the distended pelvis and urine escaped under pressure. An attempt was then made to pass a small catheter along the ureter, but it would not pass through the uretero-pelvic junction. The child recovered, and it was only later on, when I became more interested in these cases, that I realized I had missed a good opportunity of producing conclusive evidence of the occurrence of a stone in one of these cases.

Dr. Edwards says he can offer no suggestion as to why ureteric catheterization is so often prevented by œdema; neither could anyone else, because œdema *per se* would not prevent ureteric catheterization. He admits in his commentary on his own case referred to above that "bilateral ureteric catheterization is practised whenever possible". In some of the cases reported, ureteric catheterization has not only been possible, but has resulted in a cure. In many of them the catheter can only be passed for several inches, as in Barnett and Schlink's case referred to above. Dr. Kenneth Addison has informed me that in one of his cases, treated by the late Dr. Lee Brown, a cure was effected by passing ureteral catheters as far as they would go, and then injecting fluid under pressure. He stated that Dr. Lee Brown told him that he generally found this method effective. What other effect could such treatment have than the removal of a mechanical block such as a calculus? I agree with Dr. Edwards about the diagnosis of stones by pyelography, but the case referred to by me occurred before the introduction of this method, and was only mentioned in my paper to stress the importance of radio-translucency of uric acid stones.

Dr. Edwards suggests that dehydration and toxæmia are in some way associated with the œdema which he has found in the ureters in these cases. I agree about dehydration, and referred to this in my article, but I suggest that the effect of this would be to cause a deposition of crystals in the concentrated urine, and these crystals may sometimes be so abundant as to lead to their agglomeration and the formation of small calculi. I most emphatically disagree, however, that dehydration can be associated with œdema anywhere, unless the œdema is a purely local condition. The fact that Dr. Edwards found only œdema of the ureters *post mortem* in one of these cases only goes to prove that some local condition was the cause of it, and does not prove that an agglomeration of crystals was not present before death. With the death of the patient the tone of the musculature of the ureters would disappear and the emptying of the previously distended kidney pelvis would clear away the obstruction.

It may not be that a dense calculus is present in these cases, but I feel convinced that the block in all of them is a mechanical one, and is due to an agglomeration of crystals, commonly called calculi, and consisting of either uric acid oxalates or, in those cases following the administration of sulphanilamide, of an acetylated compound of the drug.

Recently I have seen another case of so-called anuria following the removal of an appendix in a child of four years of age. On the fifth day after the operation he passed only one ounce of urine, on the sixth day no urine at all, and on the seventh one-quarter of an ounce. The small quantity of urine passed on these occasions was heavily blood stained, and the first specimen contained abundant uric acid crystals. This child recovered after intravenous therapy with saline and 20% glucose. The explanation of all the signs and symptoms in these cases, and the comprehension of all the effective methods of treatment which have been employed, are perfectly clear, on the assumption that there is a calculus blocking both ureters. It is quite unnecessary, therefore, to bring forward such a condition as œdema of the ureters as a cause for this condition. I feel sure that we are going to meet with many more of these cases now that sulphanilamide is being used so extensively.

Yours, etc.,

141, Macquarie Street, Sydney, January 13, 1941. P. L. HIPSLEY.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Act of* 1918, of Tasmania, as duly qualified medical practitioners:

Whyte, Alfred Moffatt, M.B., B.S., 1939 (Univ. Melbourne), Penguin.

Sweetman, Keith Franklin Drysdale, M.B., B.S., 1937 (Univ. Melbourne), Rosebery.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Act of* 1939, of Queensland, as duly qualified medical practitioners:

Adsett, Benjamin Neil, M.B., B.S., 1940 (Univ. Queensland), 386, Annerley Road, Annerley, S.3.
Burnett, Francis Baron, M.B., B.S., 1940 (Univ. Queensland), Emmanuel College, Wickham Terrace.
Crawford, John Hamilton, M.B., B.S., 1940 (Univ. Queensland), 242, Gladstone Road, South Brisbane.
Donnan, Meredith Gordon Francis, M.B., B.S., 1939 (Univ. Sydney), General Hospital, Brisbane.
Douglas, Robert Andrew, M.B., B.S., 1939 (Univ. Melbourne), Seventh Field Ambulance, Enoggera.
Exton, William Dunham, M.B., B.S., 1940 (Univ. Queensland), 27, Jordan Terrace, Bowen Hills.
Fox, Henry Charles, M.B., B.S., 1940 (Univ. Queensland), Galatea Street, Charleville.
Gibson, John Bertram Gilchrist, M.B., B.S., 1940 (Univ. Queensland), Kingsley Hotel, George Street, Brisbane.
Grimmett, Lansell Leonard, M.B., B.S., 1940 (Univ. Queensland), Sherwood Road, Sherwood.
Hall, Noel Lyster, M.B., B.S., 1940 (Univ. Queensland), General Hospital, Brisbane.
Hemming, George Ratcliffe, M.B., B.S., 1940 (Univ. Queensland), Fleming Road, Herston.
Hawkins, Gordon Kenneth, M.B., B.S., 1940 (Univ. Queensland), Park Road, Yeerongpilly.
Hildebrand, Gregory Perry, M.B., B.S., 1940 (Univ. Queensland), Rosary Crescent, Highgate Hill.
Isles, Alexander Dennis, M.B., B.S., 1940 (Univ. Queensland), Dalkeith, McLeod Street, Herston.
Kingston, Clive William, M.B., B.S., 1940 (Univ. Queensland), General Hospital, Brisbane.
Mankin, Winifred Roby, M.B., B.S., 1939 (Univ. Sydney), General Hospital, Brisbane.
Matenson, Maurice, M.B., B.S., 1915 (Univ. Melbourne), 140, Millswyn Street, South Yarra, Melbourne.
Moreline, John Allan, M.B., B.S., 1940 (Univ. Melbourne), District Hospital, Cairns.
Macleod, Pam, M.B., B.S., 1940 (Univ. Queensland), General Hospital, Brisbane.

The Royal Australasian College of Physicians.

EXAMINATION FOR MEMBERSHIP.

INTENDING candidates for the forthcoming examination for membership of the Royal Australasian College of Physicians are reminded that applications should be in the hands of the Acting Honorary Secretary of the College, 145, Macquarie Street, Sydney, not later than February 8, 1941.

The examination will be held on the following dates:

Written examination (capital cities): March 8, 1941.
Clinical examination (Melbourne): March 29, 1941.
Clinical examination (Sydney): April 2 and 3, 1941.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Barr, Norman Duffin, M.B., B.S., 1939 (Univ. Sydney), 20, Clifford Avenue, Manly.
Gardiner, Ian Donald Russell, M.B., B.S., 1940 (Univ. Sydney), 228, Queen Street, Ashfield.

Medical Appointments.

Dr. L. A. Langley has been appointed Medical Officer in the Department of Mental Hospitals of New South Wales.

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Dr. A. M. Macdonald has been appointed Government Medical Officer at Clermont, Queensland.

Books Received.

"Mechanisms of Biological Oxidations", by D. E. Green; 1940. Cambridge: University Press. Melbourne: G. Jaboor. Demy 8vo, pp. 181, with diagrams. Price: 12s. 6d. net.

"Surgery of Modern Warfare", edited by Hamilton Bailey, F.R.C.S.; 1940. Edinburgh: E. and S. Livingstone. Super royal 8vo, pp. 164, with illustrations. Price: 12s. 6d. net.

Diary for the Month.

JAN. 31.—Tasmanian Branch, B.M.A.: Council.
FEB. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
FEB. 5.—Western Australian Branch, B.M.A.: Council.
FEB. 5.—Victorian Branch, B.M.A.: Branch.
FEB. 6.—South Australian Branch, B.M.A.: Council.
FEB. 7.—Queensland Branch, B.M.A.: Branch.
FEB. 11.—Tasmanian Branch, B.M.A.: Branch.
FEB. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 14.—Queensland Branch, B.M.A.: Council.
FEB. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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